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VOLUME LIV NUMBER 5

BULLETIN of the JOHNS HOPKINS HOSPITAL

THE PUBLICATION OF THE MEDICAL SCHOOL AND HOSPITAL
SUPPORTED BY THE DE LAMAR FUND OF THE JOHNS HOPKINS UNIVERSITY

May, 1934

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PUBLISHED MONTHLY
THE JOHNS HOPKINS PRESS, Baltimore, Maryland
Made in United States of America

BULLETIN
OF
THE JOHNS HOPKINS HOSPITAL

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Twenty-five reprints, without covers, of articles will be furnished to contributors free of cost. An order slip for additional reprints, with a table showing cost, is sent with proof.

Correspondence concerning business matters should be sent to The Johns Hopkins Press, Baltimore, Maryland, U. S. A.

This periodical is issued monthly. Two volumes a year are issued, each consisting of approximately 400 pages, and beginning in January and July, respectively.

Subscription price. In the United States and its possessions $6.00 per year. In Canada $6.25 per year; in foreign countries $6.50 per year. Prices for back volumes and back single numbers on application. Single numbers of the current volume, when available, $.75. Prices are net, postpaid.

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Agents for the British Empire, except Canada: Baillière, Tindall & Cox, 8 Henrietta St., Covent Garden, W.C. 2, London.

THE JOHNS HOPKINS PRESS
BALTIMORE, MARYLAND, U. S. A.

Entered as second-class matter at the Baltimore, Md., Postoffice. Acceptance for mailing at special rate of postage provided for in Section 1103, act of October 3, 1917. Authorized on July 3, 1918

OBSERVATIONS ON THE PATHOLOGY OF HIGH-TONE DEAFNESS

SAMUEL J. CROWE, STACY R. GUILD AND LEROY M. POLVOGT

From the Otological Research Laboratory, The Johns Hopkins University

Submitted for publication December 13, 1933

INTRODUCTION

Our main object is to establish a better basis for the intelligent interpretation of the clinical tests of hearing. The first essential is a more exact knowledge of the physiology and pathology of the organ of hearing—by this term we mean the central auditory pathways as well as the parts in the temporal bone (the middle and inner ear). Animal experimentation, and especially that done with the method of Wever and Bray, promises to yield much valuable information; the conclusions drawn from animal experiments alone, however, cannot explain the disorders of the human ear.

A large percentage of hospital patients have some type of organic lesion in the middle or inner ear, and correlation of functional tests of hearing with the exact location and extent of these organic lesions constitutes a method of physiological experimentation. The temporal bones removed at autopsy at any hospital may be utilized for such studies provided accurate hearing tests have been made a short time before death.

Our investigations on the human auditory apparatus have been limited to the structures in the temporal bone. For our purposes the determination of the exact location and extent of the cochlear lesions is most important; this requires a detailed examination of the serial sections and the making of a graphic reconstruction of each cochlea studied. When all these data are assembled and correlated with the functional tests we have a basis of facts for an interpretation of clinical tests of hearing; also, we have some information regarding tone localization in the cochlea. In addition, such material is valuable for study of the etiology of the several forms of impairment of hearing, or at least their relation to vascular disease, syphilis and other systemic

disorders. The temporal bones all come from hospitalized patients whose clinical records, laboratory tests and autopsy findings, are available for correlation with their temporal-bone lesions and their hearing defects.

Before attempting to analyze the lesions found in ears from patients with the more complicated types of deafness it is necessary first to become thoroughly familiar with the common cochlear lesions and learn how to evaluate them in terms of functional impairment. Therefore this study is limited to the temporal bones from patients whose hearing tests indicated that the lesion was chiefly in the cochlea. All cases have been included whose records show that the hearing was good for low tones and impaired for high tones alone. Histological studies show that the ears from patients with this type of hearing defect more commonly have lesions limited to the inner ear than the ears from patients with impaired hearing for all tones.

For such a study a large control group is necessary, since many ears from patients with good hearing for all tones have certain cochlear and middle-ear lesions. Histologically, the middle and inner ear lesions found in some ears with good hearing are very similar to those found in ears with impaired hearing: they differ only in extent and location. It would only lead to confusion to make interpretations regarding the functional significance of the lesions found in ears with impaired hearing without a knowledge of the extent and location of the lesions that may be present in ears with good hearing.

A group of cases has been encountered in this study in which no lesion found in the middle or inner ear adequately explains the defect in hearing. This emphasizes the need of also studying the central auditory pathways.

THE MATERIAL

It should be emphasized again that, in this attempt to correlate functional hearing tests with histologic changes, our observations are limited to that part of the auditory apparatus which lies within the temporal bone. The material for a study of the central auditory pathways is also available, but our funds are insufficient for the preparation of serial sections of both the temporal bone and the brain in any large number of cases. In this publication on the relation of certain

pathological lesions to impaired hearing, we have tried to simplify the problem by limiting the study to patients with normal hearing for low tones and defective hearing for the high tones alone.

So many different structures in the middle and inner ear take part in the transmission and perception of sound that the first essential for such a study is an abundance of material. Since 1924 we have collected and sectioned serially more than 1400 temporal bones, and for nearly 800 of these patients the hearing had been tested with the audiometer. The pathological department of a large hospital provides ideal facilities for the collection of material for an investigation of this kind. With the coöperation of the medical, surgical and other clinical departments, we test the hearing of patients whose general condition makes it seem likely that they will not recover. By this method of selection large numbers of temporal bones are secured from patients whose hearing is normal; this material, when sectioned, is of great value as a control and check on histologic artefacts and postmortal changes and, in addition, demonstrates the fact that certain pathologic changes may be present in the middle and inner ear with no associated impairment of hearing. Many of these patients, however, do have impaired hearing; all stages of otosclerosis (1), and many types and degrees of deafness from other causes (2) are found. For the study of the etiology of deafness and the relation of the ear to general systemic disorders, the hospital history, the clinical laboratory tests and the complete autopsy records are available for correlation with the hearing and vestibular tests and with the histologic sections of the temporal bones.

Our functional tests of the auditory and vestibular apparatus are often incomplete because the subjects are seriously ill, but every precaution is taken to insure the accuracy of the tests that can be made without unduly disturbing or tiring the patient. It would not be possible to correlate with accuracy the functional tests and structural changes if we had to depend entirely on the voice, the watch and the tuning-forks in order to determine the hearing acuity. The results of such tests may be incorrect as a result of subjective factors, so the first test is always made with the electric audiometer. Two specially trained, full-time assistants make all audiometer tests. If the patient is too sick to be moved, the test is made on the ward, but whenever

possible the sound-proof room is used. A detailed otoscopic examination and the usual tuning-fork tests, and, in addition, functional tests of the vestibular apparatus, are made if the condition of the patient permits. The audiometer is absolutely essential for our purpose because the test with this instrument can be made in such a way as to be free from subjective impressions, the examination can be made without fatigue or exertion for the patient and the threshold of acuity for all tones, including the higher frequencies, is accurately determined.

The audiograms of the 79 ears used in this investigation are similar in that the acuity recorded for the low tones is within the limits of normal, while the acuity for the high tones is either impaired or lost. In this way we simplify our problem. The correlations of function and pathology would be inaccurate and confusing, however, if they were based solely on the similarity of the audiograms. A number of conditions must be fulfilled when selecting the material for a study of this kind, and it was necessary for us to discard more than 100 ears because they did not comply with all of the following points: (1) the audiogram must show that the hearing is impaired for all tones higher than the one at which the loss begins. This excludes the "tone dips." (2) The high-tone loss or impairment must include the tone c^6 (8192 d.v.). The audiograms in which the loss or impairment is limited to one or more of the highest frequencies produced by the 1-A Western Electric Co. audiometer (10,321, 13,004 and 16,384 d.v.) have been discarded. This was necessary because many of our cases were tested with the 2-A audiometer whose upper limit is 8192 d.v. (3) The notation made at the time the patient was tested must indicate that he or she was coöperative and that the examiner regarded the audiogram as a reliable record of the hearing. (4) The time interval between the hearing test and the removal of the temporal bones is important. Nearly three-fourths (73 per cent) of the 79 ears selected were removed within six weeks after the hearing had been examined. For 24 ears the time interval was within 8 days; for 22 between 9 days and four weeks; 12 of the temporal bones were removed during the fifth or sixth week; for 15 ears the interval was from $2\frac{1}{2}$ to $8\frac{1}{2}$ months and for the remaining 6 from 10 to 17 months. (5) The time interval between the death of the patient and the removal of the temporal

bones is also important. Many cases that fulfilled all other conditions had to be excluded because post-mortal autolysis of the organ of Corti was so advanced that no definite statement could be made as to its condition during life. (6) The histologic sections of the control cases with normal hearing, as well as the cases with impaired hearing, must be good enough to permit a positive statement as to whether the finer structures in the cochlea are normal or pathologic.

When assembling for this publication all available material in the laboratory we find that 188 temporal bones have been sectioned whose audiograms fulfill the first two conditions; application of the other four requirements, however, reduces the number to 79. This is a comparatively limited material, but the careful selection enhances the accuracy of our conclusions.

The technique used in the preparation of the histologic sections was uniform for all of the 79 temporal bones selected, with but four exceptions. The technique for these four differs from that outlined below in that two were fixed in Müller-formol and two in Zenker-formol. The differentiation between organic lesions and the artefacts of post-mortal autolysis and method of preparation is based on our study of the serial sections from 200 tested cases with good hearing that were prepared with the same technique.

Perfect preservation of *all* tissues and cells in human temporal bones is impossible even if the autopsy is obtained within two or three hours after death. A method must be used, therefore, that gives the best average preservation of all tissues, rather than one which is excellent for a few structures and poor for others. The technique outlined below is the result of experiments with various methods that extended over a period of four years, and the use of several hundred temporal bones. This method gives better average results for all tissues than any of the others we have tried.

(1) The temporal bones are removed from the skull with a rotary saw in order to avoid fractures and the concussion effects of chiselling.

(2) The gases in solution are removed from the water used in preparing the fixing fluid, the decalcifying fluid and the sodium sulphate solution; also from the diluted alcohols. It is especially important that the running water, used for washing the specimen after removal from the sodium sulphate bath, be properly "degassed" (3). This is accomplished by having a large tank fitted with steam

coils in which the water is constantly boiling. The boiling water passes through cooling coils and is distributed to the various jars in which the specimens are suspended on threads.

(3) For fixation the two temporal bones are suspended in at least 400 cc. of a 20 per cent solution of formalin for the first 24 hours. They are then transferred to an equal amount of a 10 per cent formalin solution, which is changed daily for the first three days.

(4) At this stage the specimen is cut down to the proper size and shape with a rotary saw, and notes are made on the appearance of the tympanic membrane, the extent of the pneumatization, *etc.*

(5) For decalcification, a 1 per cent solution of nitric acid in 10 per cent formalin is used. The specimens are suspended at least one inch below the surface in 400 cc. of fluid. The fluid is changed daily. The time required for decalcification varies even in the same age group, and a separate jar is used, therefore, for each pair of temporal bones. We have found that a chemical test for the presence of calcium in the decalcifying fluid is more satisfactory than the use of a needle or x-rays to determine the end-point of decalcification. The total time required varies from 22 to 35 days.

(6) When decalcification is complete, each pair of temporal bones is transferred to a 5 per cent solution of sodium sulphate for two days, with one renewal of fluid. This improves the staining properties of the tissues.

(7) The specimen is then washed for 48 hours in "degassed" running water in order to remove the sodium sulphate as well as any remaining acid.

(8) The strengths of the graded alcohols used in dehydration are: 35, 50, 60, 70, 80, 90, 95 (two changes), "used" absolute and absolute (two changes).

(9) Celloidin infiltration and embedding are used. For satisfactory results with temporal bones, much longer times must be allowed for each stage than with soft tissues of a comparable size. We have found that three weeks in 4 per cent celloidin and five weeks each in 8 per cent and 12 per cent are minimum times. After this the specimens are placed in 16 per cent celloidin in Stender dishes; the rate of evaporation is so controlled that about two months are required to reach a moderately firm consistency. The celloidin is then hardened with chloroform vapor, followed by 80 per cent alcohol. The entire block is sectioned serially at 24 micra. Every tenth section is stained with Ehrlich's hematoxylin and eosin and mounted as a "tracer" series. The intermediate sections are stored in 80 per cent alcohol in known sequence and as needed are prepared with the special stains.

THE METHOD OF STUDY

The first step in the study of the 79 ears thus selected is to record as accurately as possible the location and extent of the cochlear lesions. The method of graphic reconstruction described by Guild (4) in 1921,

has been used for this purpose. The completed graph may be regarded as an ortho-projection diagram of the parts shown, and assembles on one chart the results of many hours of study with the high power of the microscope. Normal structures, as well as the pathologic changes, are recorded for each tenth section. A chart of this kind is much more satisfactory than written descriptions for the grouping and comparison of cochlear lesions. Modifications of this method have been used in two previous publications from this laboratory (5, 6).

The graph is plotted on a large sheet of ruled paper on which each horizontal space is regarded as representing one of the serial sections, and is numbered accordingly. In vertical series there are five sections in which the line of contact between the heads of the outer and the inner pillar cells of the organ of Corti is cut tangentially.[1] The mid-points of the spaces corresponding to these five sections are joined by a continuous line made up of four semicircles. The semicircles represent the "axis of the organ of Corti" of the lower apical, upper middle, lower middle and upper basal turns.

The graphic representation of the lower basal turn consists of two segments: (1) a 90° circular arc connecting the end of the semicircle for the upper basal turn and a point representing the greatest "bulge" on the lower basal turn, and (2) an arc with a longer radius connecting this latter point and the point representing the extreme basal end.

The location of these two points is determined as follows: The section with the organ of Corti cut radially is at the level of the greatest "bulge," which is usually somewhat posterior to the mid-modiolar region. In this section the actual distance between the pillar cell junctions in the lower middle and lower basal turns is measured with a micrometer ocular. The "bulge" point of the lower basal turn is thus located on the ruled paper. The position for the point representing the extreme basal end cannot be determined by direct measurements, since in vertical series other turns of the cochlea are not present in the section containing the basal end. Therefore the level of the extreme basal end with respect to the tangents of the lower middle and lower apical turns was determined in a group of cochleae cut in the horizontal plane, and the average of these observations used for

[1] There are only four such tangential cuts when the apical turn is of the short variety.

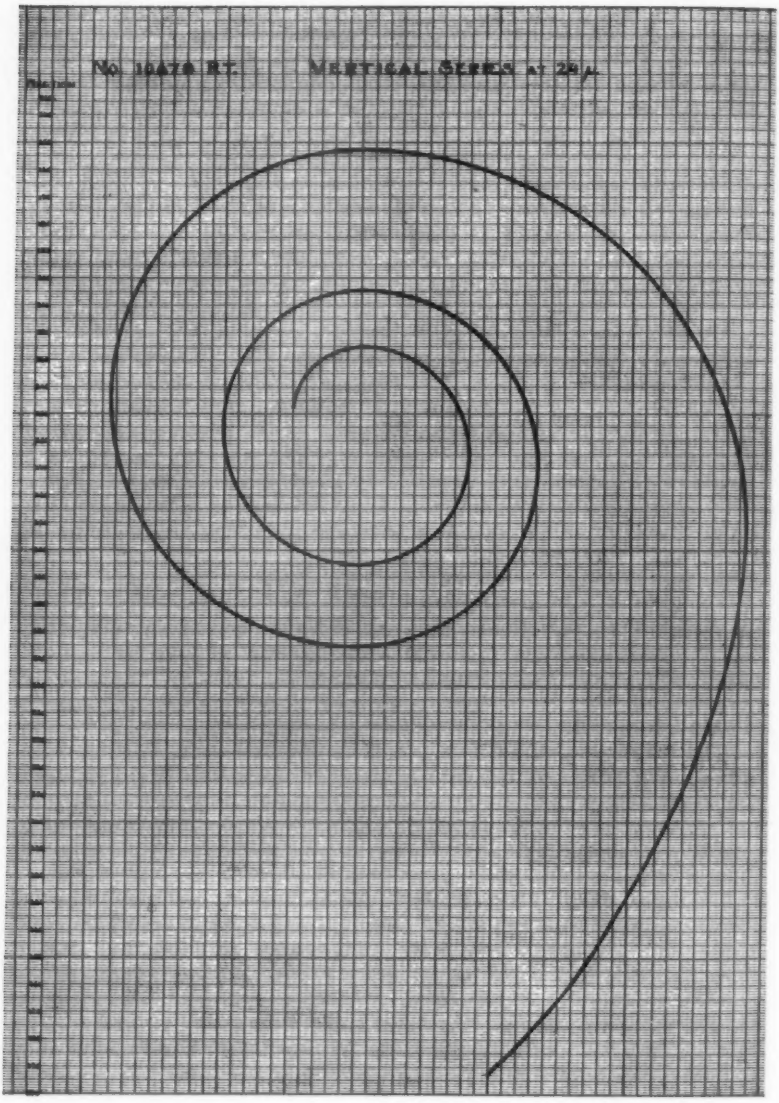


FIG. 1. The graphic reconstruction of the "base line" for a particular cochlea. (See text, p. 321.)

locating the basal end point on the graphs of the cochleae cut in the vertical plane. The necessary points for the upper apical turn are determined by direct measurements of the sections.

The entire base line for a particular cochlea, as reconstructed by this method, is shown in fig. 1.[2] The magnification is the ratio of the width of one space to the average thickness of one section. Symbols grouped about such base lines record for each cochlea the conditions observed in the nerve fibres in that part of the osseous spiral lamina under the limbus, the organ of Corti, the external sulcus cells and the stria vascularis. These symbols are placed in correct relative position to each other, but are not made in true proportion to the actual size of the structures they represent. Instead, they are so designed that they may be easily identified when the large chart is reduced to publication size. The conditions observed in every tenth section of the series are recorded on the graph; we have found this interval sufficiently small to determine the extent of the lesions present, and it avoids undue crowding of the symbols.

In our cases, with good hearing for low tones and impaired hearing for high tones, the nerve, organ of Corti and external sulcus cell lesions are invariably limited to the basal turns (the only exception is an occasional case with a questionable atrophy of the nerve to the lower part of the middle coil). For this reason the graphic reconstruction spirals record the condition of these various structures for the basal turns alone. The stria vascularis may be atrophic in limited areas in the apical and middle as well as in the basal turns. This finding, however, is not recorded in the graphs and is not regarded as significant because the lesions of this structure in some of our control cases, with good hearing for all tones, have a similar distribution.

[2] The use of such a series of arcs with abrupt changes in curvature to represent a structure whose curvature changes continuously from the basal to the apical end introduces an error which for most regions does not exceed ± 5 per cent. That part of the basal end of the basal turn (pars vestibularis), which deviates markedly from the general plane of the rest of this turn, is not well represented by this method of graphic reconstruction from serial sections in the vertical plane. Since we have been unable, however, to demonstrate any relationship between the pathologic lesions observed in this region and the acuity of hearing for the tones used for testing, the large error in plotting the extreme basal end is of no importance for our present problem.

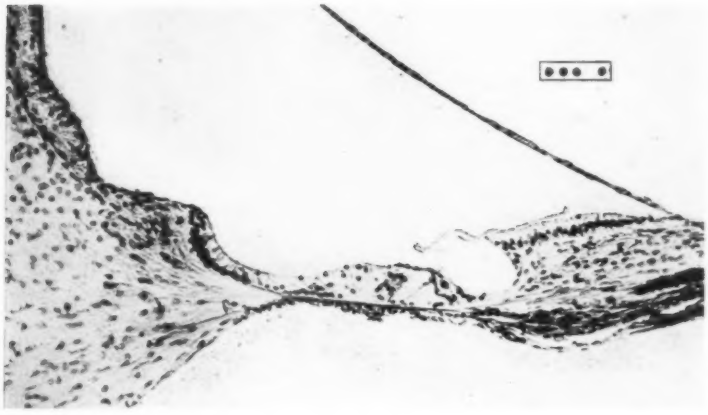


FIG. 2. Normal organ of Corti and the symbol used to represent it in the spiral graphic reconstruction charts. Each hair cell is represented by a dot. The external sulcus cells and the nerve are normal. (Figs. 2 to 6 are all from about the same region of the lower basal turn; they are reproduced at a magnification of 150 diameters.)

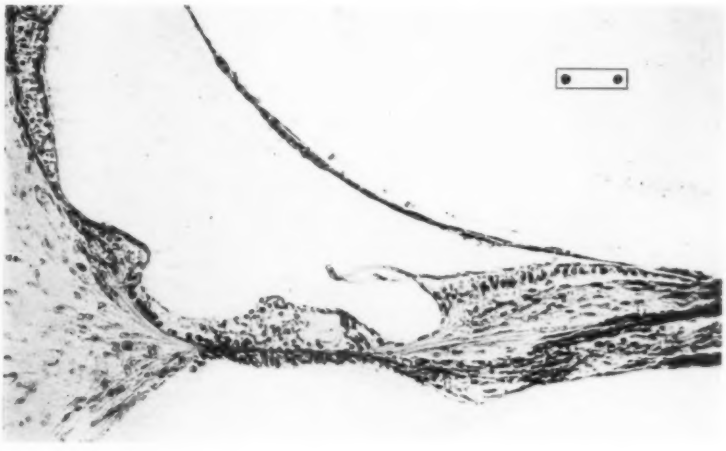


FIG. 3. The first and second of the outer hair cells are missing in this organ of Corti; the symbol shows how this condition is represented in reconstruction. The external sulcus region has less than the normal number of nuclei.

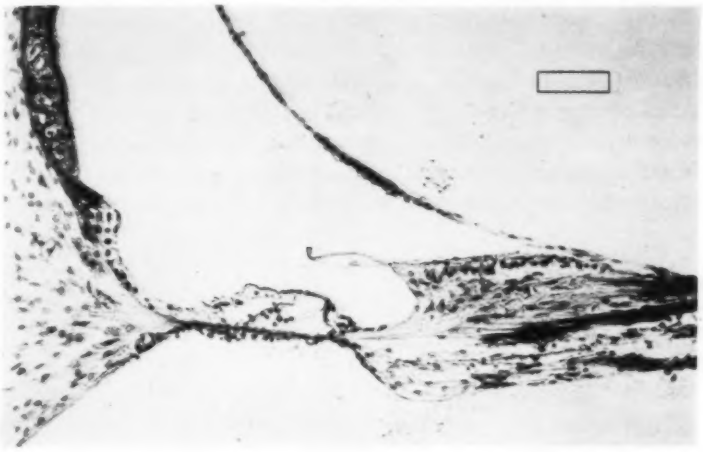


FIG. 4. The supporting cells of the organ of Corti are present, but there are no hair cells; the symbol represents this empty "framework." Comparison with fig. 2 shows that there is definite partial atrophy of external sulcus cells and nerve.

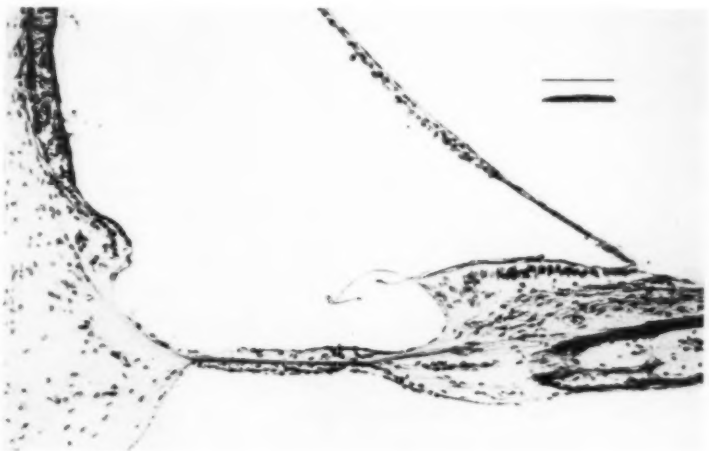


FIG. 5. This condition of the organ of Corti is termed "almost total atrophy;" the black area placed on the lower line of the symbol in the reconstructions records the size of the remains of the supporting cells relative to the normal.

The symbols used to indicate the several conditions observed in the organ of Corti are shown, with photomicrographs of typical examples, in figs. 2 to 6. The general plan is to represent the supporting framework of the organ of Corti by a quadrilateral "box," and the hair cells by dots within this outline. Thus a radial section of a normal organ of Corti is indicated by a "box" with four large dots (fig. 2); these dots are so placed with reference to the base line representing the heads of

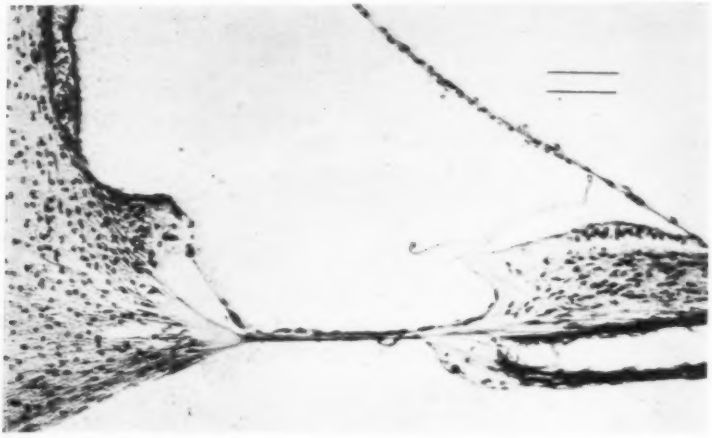


FIG. 6. This condition, total atrophy of the organ of Corti, is recorded on the spiral reconstruction charts by two parallel lines. This section also shows total atrophy of the external sulcus cells and the nerve. (The detachment of the tectorial membrane is an artefact, as is also the "lifting" of the squamous cells which cover the external sulcus region.)

the pillar cells as to be correct for the relative positions of inner and outer hair cells. Small dots are used to represent the hair cells whose appearance in the section differs from the normal. From the experience gained by studying the sections of many cochleae with known good hearing we feel quite certain that in most instances where small dots are used the altered appearance is due solely to autolytic or postmortal changes.

Absence of dots in the expected positions in a "box" indicates that the corresponding hair cells are not present in the section, but the

supporting structures—pillar cells, Deiters' cells, inner sustentacular cells and the cells of Hensen—are still present and the general form of the organ of Corti is retained (figs. 3 and 4). We believe that in most instances the absence of hair cells is pathologic and is not due to changes after the death of the patient. The strongest evidence for this view is the fact that, in each of these questionable cases, the organ of Corti is normal in appearance in other parts of the cochlea. Also the sections with one or more hair cells missing usually adjoin a region of total atrophy of the organ of Corti.

When the basilar membrane is covered by a layer of columnar or cuboidal epithelium, and the characteristic specialized elements of the organ of Corti are not recognizable, the symbol used is shown in fig. 5. The distance between the two horizontal lines, which are not joined at the ends to form a "box," is regarded as representing the height of the normal organ of Corti, and the relative size of the "hillock" of epithelial cells present is indicated by the black area on the lower line. We use the term "almost total atrophy" for this condition; no neuro-epithelial cells are recognizable, and from the standpoint of auditory function it is doubtless equivalent to a total atrophy of the organ of Corti.

Certain methods of histologic preparation frequently cause so much shrinkage of even a normal organ of Corti that, when seen with the lower magnifications, there is a superficial resemblance to the "almost total atrophy" just described. With higher magnification, however, it is evident that the two conditions are entirely different. The otological literature contains many illustrations in which various degrees of shrinkage are described as partial atrophy of the organ of Corti, and the explanations of the impaired hearing are thus based on histologic artefacts. Because of this literature and the great difference it might make in the interpretation and understanding of our data by others, we wish at this time to point out that we do not regard as pathologic any organ of Corti that in section appears smaller than is usual in that region, provided all of the characteristic cellular elements can be recognized with the higher powers of the microscope. Our experience with numerous methods of histologic preparation has convinced us that this shrunken appearance of the organ of Corti is always an artefact due to the technique and is of no functional significance.

Total atrophy of the organ of Corti is graphically represented by two parallel lines not joined at the ends (fig. 6). The basilar membrane is covered only by a layer of squamous epithelium. This condition is easily recognized in even poorly preserved material.

Each of the three other structures—the nerve in the osseous spiral lamina under the limbus, the cells of the external sulcus and the stria

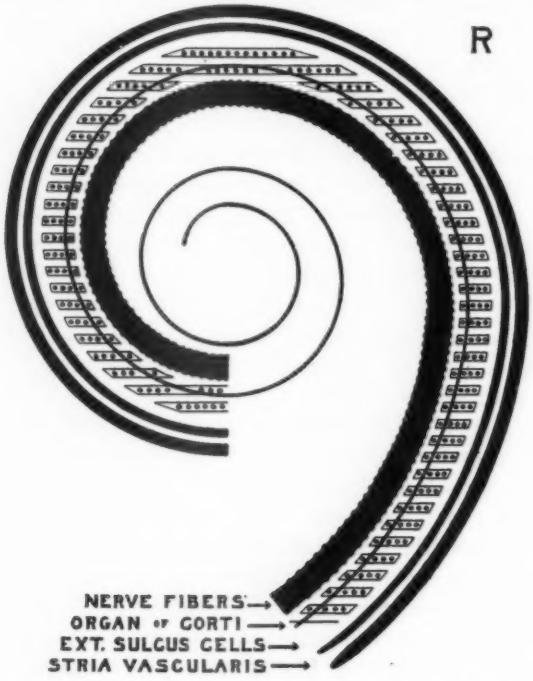


FIG. 7. The spiral graph of a cochlea in which all of the recorded structures are normal at all levels of the basal turn. (This is ear no. 11 of table 1)

vascularis—are first represented on the chart by an outlined zone, concentric with the base line, and in the proper relative position to the symbols for the organ of Corti. As each section is studied a notation is made in the proper zone as to the condition of these structures. The simple method of using solid black to represent the

structure present and leaving blank that proportion of the zone which corresponds to the degree of atrophy gives a satisfactory graphic record.

The completed charts shown in figs. 7 to 9 demonstrate clearly the manner in which the various symbols are used in combination.

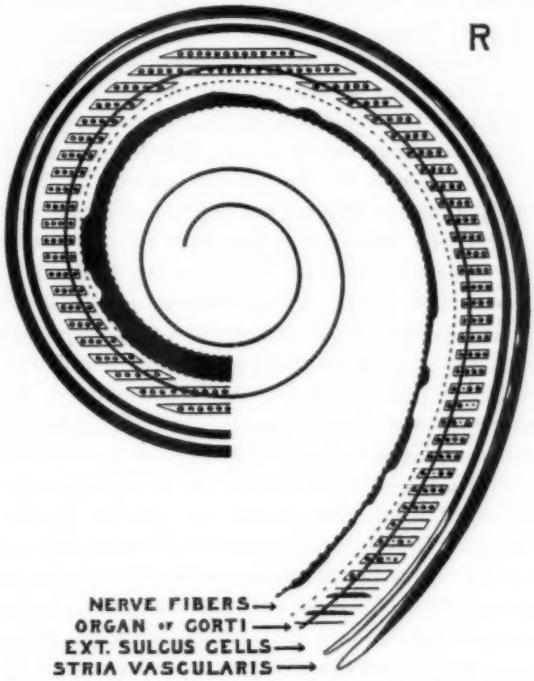


FIG. 8. The spiral graph of a cochlea in which the principal lesion is a partial atrophy of the nerve in the entire lower and more than half of the upper basal turn. (This is ear no. 15 of table 2.)

The first of these charts (fig. 7) is that of a cochlea in which all of the structures indicated are normal at all levels. Accordingly, the chart shows each of the three zones completely filled in with black and each symbol for the organ of Corti is a "box" with large dots representing the inner and the outer hair cells.

The second chart (fig. 8) records a partial atrophy of the nerve in the osseous spiral lamina of the entire lower basal turn and in more than half of the upper basal turn. At each level of the zone labelled "nerve fibres" the fraction of the zone which is black indicates the ratio between the amount of nerve present in the corresponding section and the amount that we regard as normal for that region. In young adults with good hearing for all tones there is a marked difference in the amount of nerve in the several regions of the cochlea. There is more nerve in the upper basal and middle turns than elsewhere, and the amount diminishes rapidly as the basal end is approached. To show the absolute amount of nerve and its relation to the normal for each level would complicate the construction of the chart and add confusion to our present attempt to correlate structure and auditory function. Therefore, the zone on the chart that represents the normal amount of nerve is uniform in width, and it should be kept in mind that our charts show only the relative amounts. Each judgment as to the relative amount of nerve in a section, proportionate to normal for the level, is based on the personal experience of the authors.

Relative innervation of areas of the organ of Corti, as measured by the method of counting ganglion cells in the adjacent parts of the spiral ganglion and comparing with the normal average, is known for 28 ears of this material. The figures for 23 of these are from the unpublished work of Dr. Mary Hardy (6), and for the other five are from the paper by Guild, Crowe, Bunch and Polvogt (5) in 1931. For these 28 ears the correlation of hearing acuity with the amount of nerve in the peripheral part of the osseous spiral lamina is much better than with the ganglion cell counts; therefore, we have not proceeded with the immense task of counting the ganglion cells in the other 51 ears. The figures for the total number of ganglion cells, and for the number per millimeter of length of the organ of Corti in the lower and the upper parts of the basal turn are included in our tables. Possible reasons for the discrepancies in some individuals between ganglion cell data and hearing acuity will be discussed in Dr. Hardy's paper (*q.v.*).

The ideal method for the nerve record would be to count the number of fibers leaving the osseous spiral lamina to enter the organ of Corti; since most of the nerves in this region are sectioned obliquely or longi-

tudinally, this is a very difficult procedure. We have attempted to perfect a stain which would make this possible, but without success.

The third chart (fig. 9) is that of a cochlea with total atrophy of the organ of Corti, the nerve and the external sulcus cells in the lower

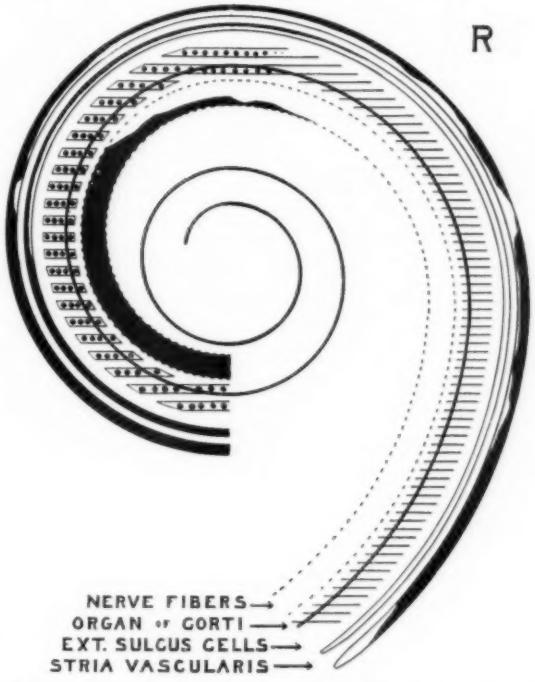


FIG. 9. The spiral graph of a cochlea with total atrophy of the nerve, organ of Corti and external sulcus cells in the lower basal turn. (This is ear no. 69 of table 5.) Figs. 7, 8 and 9 show how the method of using the symbols in combination gives a visual record of the location and amount of atrophy in a cochlea.

basal turn (fig. 10). The transition from total atrophy to a normal appearance is in this cochlea abrupt for the organ of Corti and gradual for the nerve and external sulcus cells, as indicated in the chart. The condition of the stria vascularis varies in the different sections from normal to total atrophy.

The construction of the outlines alone for the chart of one cochlea requires several hours of work before recording the conditions observed in the individual sections, which is an even longer task. Therefore, a composite graph was made in which the base line represents the average for 68 cochleae. This outline, reproduced by photoengravure and printing in reversed images for the right and left ears, has been used in making the reconstruction graphs of all cochleae referred to in this publication. It is easy to adapt this outline to the recording of both the lower and the upper basal turns of the individual cochleae.

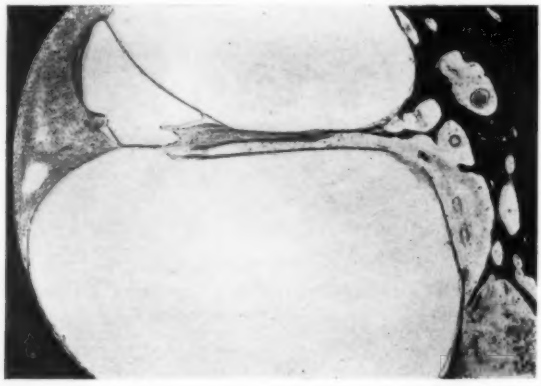


FIG. 10. Section from about the middle of the atrophic area shown in fig. 9. In this ear there is also a marked degree of atrophy of the spiral ganglion, but in some ears with a similar peripheral atrophy the number of ganglion cells is normal.

The slight loss in accuracy of proportions is more than compensated for by the ease with which many records may be compared and grouped.

Spiral diagrams of the cochlear conditions have been made in this way for each of the 79 ears with high-tone loss that meet all of the requirements for inclusion in this study.

As a control group, the spiral diagrams of 15 ears with known good hearing for all tones have been selected as representative of the range from entirely normal structure to the greatest amount of cochlear atrophy that we have found in cases with good hearing. The real

control group from which these 15 were selected numbers about 200 ears, and is the basis for our interpretation of the pathologic changes in the middle and inner ears of the group with defective hearing.

We wish to point out at this time that these diagrams are records of the best judgments of three observers as to the conditions present

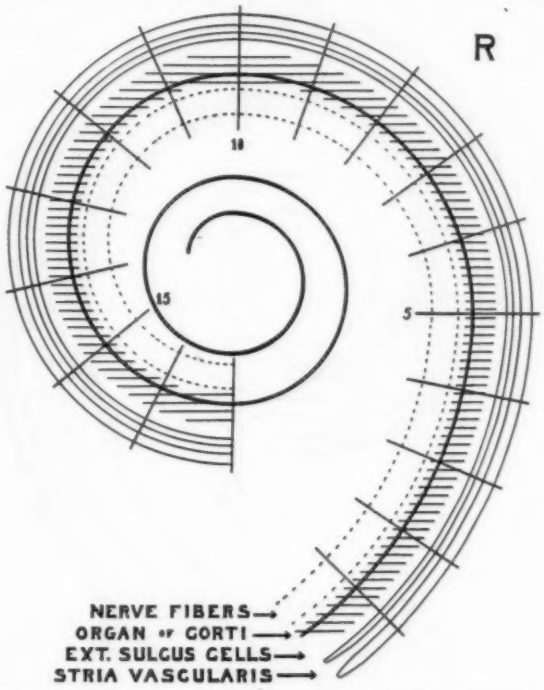


FIG. 11. The outline form of a spiral graph, marked off to show the approximate equivalents of millimeters of actual length of the organ of Corti.

in every tenth section of each series. They are not merely schematic representations of the pathological changes that, theoretically, would account for the loss of hearing.

As the next step in this study, all pertinent information from the clinical histories, the otological examinations, the general autopsy findings, the histological study of the middle ears, *etc.*, was assembled

into one large table. To this was added, in a linear form, the information contained in the spiral graphs of the cochleae. Each structure—nerve, organ of Corti, external sulcus cells and stria vascularis—is represented in the table by an equal length of a horizontal space. In other words, each structure is shown as though it were separately

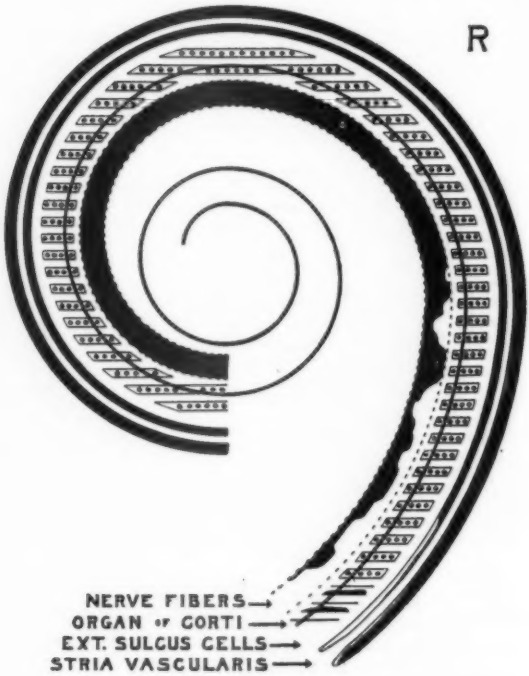


FIG. 12. (This is ear no. 43 of table 4)

FIGS. 12, 13, 14 and 15 show, respectively, three spiral graphs of cochleae and their representations in linear chart form. The accurate transfer of the spiral graphic records of cochlear lesions to the linear chart form is facilitated by superimposing the outline form shown in fig. 11 on the reconstruction graphs.

"unrolled" and transferred to the linear chart with the basal end to the left.[3] To facilitate the transfer of a record from spiral to linear form,

[3] Since the structures of the middle and apical turns are normal in this material, except as noted above (p. 323), representation of these turns is omitted in the linear charts.

an outline spiral chart with the basal turn marked off into 17 equal parts (fig. 11), each equivalent to approximately 1 mm. of actual length of the organ of Corti, is superimposed on the spiral reconstruction graph. With each space of the linear chart similarly divided, accurate transfer of the data is made possible.

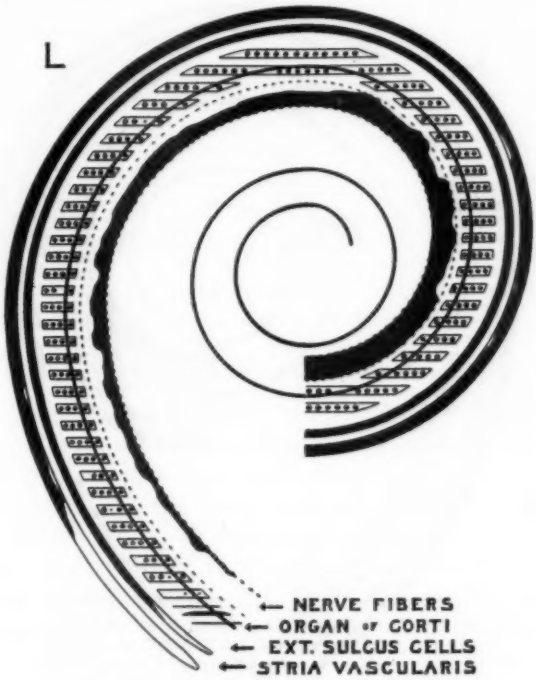


FIG. 13. (This is ear no. 13 of table 2)

The symbols for the organ of Corti in the linear table necessarily differ from those used in the spiral graphs. For the other three items the representation of atrophy and normal structure in the linear charts is the reverse of that used in the spiral graphs, because in tabulated form the use of black for atrophy brings out the facts more clearly. The proportion of each linear space filled with black records the degree of atrophy.

A continuous region of total atrophy of the organ of Corti is indicated by filling completely the corresponding part of the space with black; and isolated short areas of total atrophy by vertical black bands of appropriate width extending across the entire space. The condition of the organ of Corti termed "almost total atrophy," de-

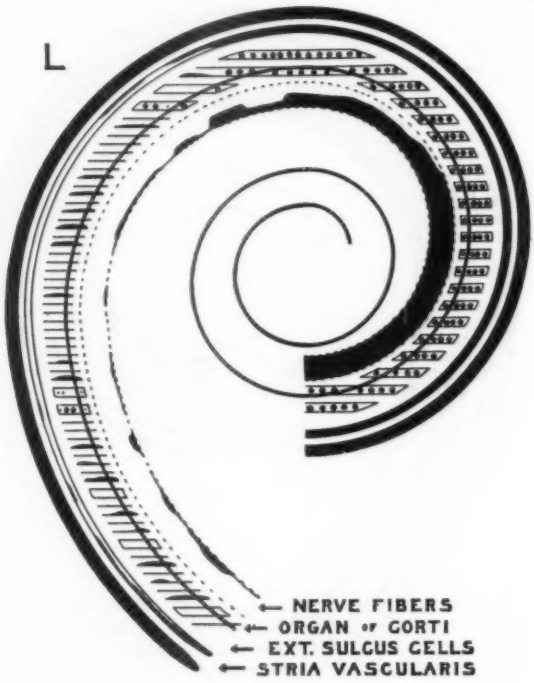


FIG. 14. (This is ear no. 56 of table 5)

scribed on page 327 and illustrated in fig. 5, is represented by a vertical band extending across three-fourths of the width of the space. When all of the hair cells are missing, but the general form of the organ of Corti is retained (p. 326 and fig. 4), the condition is indicated by black extending across one-half of the width of the space. When only a few of the hair cells are missing (p. 326 and fig. 3) the blackened area used to record the condition extends across one-fourth of the width of the

space. For the normal organ of Corti, the corresponding part of the space is left entirely blank.

To illustrate this method of recording and make it more clearly understandable, three spiral graphs and their representations in linear form are shown in figs. 12, 13, 14 and 15.

This arrangement of the cochlear data has greatly facilitated the making of comparisons, groupings and analyses of the material.

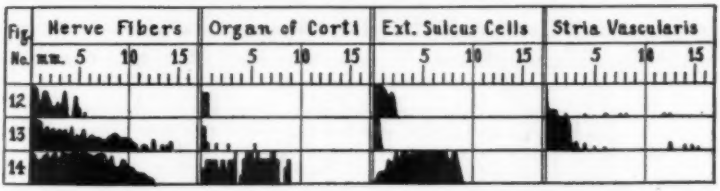


FIG. 15. (See text, p. 335, for the explanation of the symbols used to record in linear chart form the graphic records of the spiral reconstructions.)

THE LESIONS ASSOCIATED WITH HIGH-TONE LOSS

By this method of collecting and studying human temporal bones we are trying to learn something about the physiology of the ear and the pathology of this particular type of hearing loss. Every case may be regarded as an experiment on a human being since the audiogram and other functional tests of hearing are available for correlation with the lesions. The difficulty in drawing accurate conclusions arises from the fact that the lesions seen in the histologic sections are usually multiple and involve more than one structure in the middle or inner ear.

The Control Group

The control for this study consists of a group of about 200 ears with known good hearing for all tones up to and including 8192 d.v. The histologic sections of this control group have been studied by the same methods employed in the study of the ears with impaired hearing. It is rare to find all structures of the middle and inner ear entirely normal, and the lesions found in these control cases must be considered in offering any explanation for defective hearing.

The data for 15 of this control group are given in table 1. They have been selected to show the range from entirely normal structure to the greatest amount of cochlear atrophy which we have found in any case with good hearing. The audiogram and the spiral graph of one with definite cochlear pathology are presented in figs. 16 and 17.

The Middle Ear. Middle-ear lesions more severe than in any of the 15 ears selected are seen in the sections of some of the cases with good hearing. Among the more common middle-ear lesions in the control group are the following: (1) marginal and moderate diffuse thickening of the tympanic membrane; (2) small scars and calcified areas in the

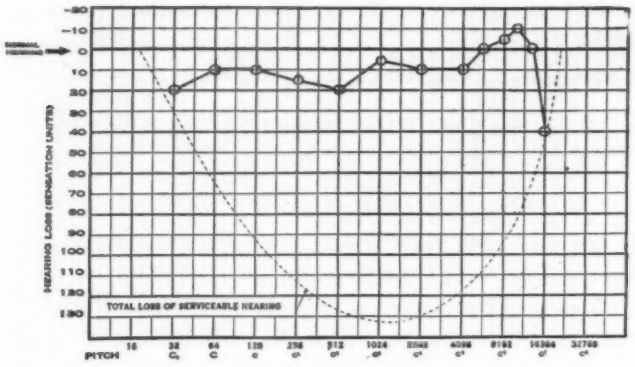


FIG. 16. Audiogram of ear no. 6 of table 1, showing good hearing for high tones. Age, 31 years.

tympanic membrane; (3) retraction of Shrapnell's membrane without the formation of much scar tissue in Prussak's space; (4) fibrous adhesion of the posterior crus of the stapes to the promontory; (5) small exostoses near the anterior margin of the oval window which do not impinge upon the foot-plate of the stapes or have fibrous adhesions to its anterior crus; (6) fibrous bands or small masses of myxomatous or other tissue in the round window niche; (7) fibrous thickening of the mucosa of the promontorial region or of the hypotympanum, and (8) thickening of bone and mucosa in the antero-superior part of the middle ear that does not, however, involve the orifice of the Eustachian tube or the ossicles. The presence of one or more of these lesions

in ears with poor hearing cannot be regarded as the cause of the impairment.

The Cochlear Nerve. The nerve supply to the lower end of the basal turn is always less than that to other parts of the cochlea and, in the

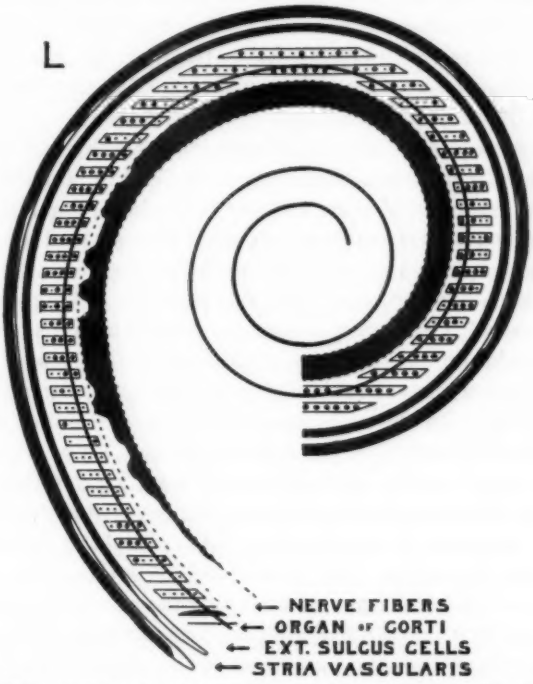


FIG. 17. This spiral graph, of the cochlea whose audiogram is shown in fig. 16, illustrates the cochlear pathology that may be present in patients with good hearing.

last 4 mm. particularly, there is a great variation in the amount of nerve in ears with similar hearing acuity for all tones up to and including 8192 d.v. The variation compatible with good hearing increases as the basal end is approached. A nerve atrophy averaging as much as 50 per cent in the lower 4 mm. of the basal turn and 20 per cent in the next 3 mm. is frequently observed. This is well illus-

trated by the amount of nerve atrophy in the ear whose audiogram and spiral reconstruction chart are shown in figs. 16 and 17. While the acuity for tones above 8192 in this patient is unusually good, the amount of nerve atrophy is greater than in others of the control group whose hearing is impaired for these higher tones. In fact there is a total absence of nerve in the last millimeter. On the other hand, we have never observed a nerve atrophy in the apical, middle or upper basal turns of cases with good hearing for all tones up to and including 8192.

The Organ of Corti. Our cases with good hearing and good histologic preservation have a normal organ of Corti at all levels with the occasional exception of some atrophy in the last 2 mm. of the basal end. Therefore, impaired hearing for the tone c^6 (8192 d.v.) cannot be explained by an end-organ lesion limited to the lower 2 millimeters.

The External Sulcus Cells. With our histologic technique the region of the external sulcus, when normal, is covered with a single layer of high cuboidal or low columnar epithelium. The cellular processes extending from the external sulcus into the spiral ligament are not differentiated with the ordinary stains. Our description of the external sulcus, therefore, is based on the appearance of the superficial cells (see figs. 2 to 6). In patients with good hearing these cells are normal in all parts of the cochlea, with the frequent exception of atrophic changes in the last 4 or 5 mm. of the basal end (table 1).

The Stria Vascularis. The stria vascularis covers at the maximum that part of the spiral ligament from near the base of the prominentia spiralis to the attachment of Reissner's membrane. This extent we regard as the normal; the proportion of this distance which the stria actually covers is indicated in our charts, and we speak of the part not covered as atrophic. There are many cases with good hearing, however, in which at several levels in the cochlea the stria does not cover this maximal surface. This raises the question as to whether atrophy is the proper term for the condition, since developmental variations may account for at least part of the apparent loss. In either case our method of recording the condition seen in the serial sections presents the facts as to the amount present. The probable function of the stria vascularis is the secretion of endolymph. This fluid in its movement along the cochlear canal (7) conveys nutrition to the hair cells

of the organ of Corti and waste products of metabolism away from them.

The total area of the stria, as averaged for the entire cochlea, is in some cases with good hearing not more than three-fourths of the possible maximum; in some of these ears there are areas as long as 2 mm. or more in which the stria is less than half its usual width. Such atrophic areas are found in the apical and middle as well as in the basal turn. It is certain that these moderate reductions in the total surface area of the stria vascularis do not affect hearing acuity. These facts must be kept in mind when considering the cases with impaired hearing.

The Basilar Membrane. Otto Mayer (8), in 1919, supported the idea that an increase in the rigidity of the basilar membrane is an important factor in the causation of presbyacusia, the impaired hearing of old age. He found the lower end of the basilar membrane always thickened and frequently ossified in the ears of 28 patients over 60 years of age. He did not find these changes in the basilar membrane of younger individuals. Many cases in our control group (table 1) have changes in the lower 2 mm. of the basilar membrane similar to those described and illustrated by Mayer. Such changes in the extreme basal end are not limited to older individuals and do not affect the hearing acuity for the tone 8192 d.v.

With this information on the histologic changes in the control group as a basis for comparison we may now analyze in a similar way the cases with good hearing for low tones but impaired hearing for high tones.

The High-Tone Loss Cases

The majority of all patients with a high-tone loss belong to one or the other of two groups. The one which we term "abrupt high-tone loss" has an approximately normal hearing acuity for all tones up to some particular frequency, and is characterized by the abrupt and marked impairment of hearing for the next higher tone and for all the yet higher tones, if they are heard at all. The audiogram of a typical example is shown in fig. 18.

The other group, termed "gradual high-tone loss," has on the average a slight or moderate decrease in hearing for the successive higher tones. Fig. 19 shows the audiogram of a typical example. It is to be

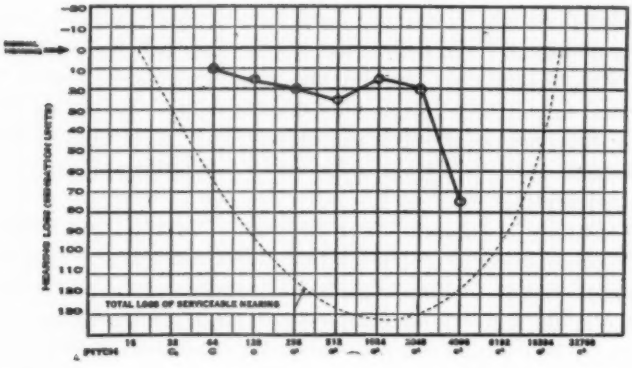


FIG. 18. Audiogram of a typical example of the "abrupt high-tone loss" group

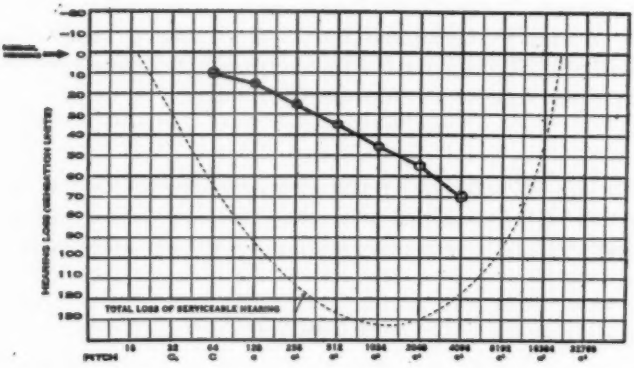


FIG. 19. Audiogram of a typical example of the "gradual high-tone loss" group

noted that the terms "abrupt" and "gradual" are used to describe only the loss of acuity for high tones at the time of examination (the audiogram), and do not carry any implication whatever as to the elapsed time during which the hearing dropped from normal to the present level.

The histologic lesions found in those ears for which we have sections and functional tests also enable us to divide the high-tone loss cases into two groups. Partial atrophy of the nerve supplying the basal turn is the most striking lesion in the "gradual high-tone loss" group, while in the "abrupt high-tone loss" group both the organ of Corti and the nerve are seriously damaged. These are the most constant lesions, but in each group there are cases with either histologic changes in other structures, particularly in the middle ear, or an absence of any lesion in the temporal bone that would explain the hearing defect. It is necessary, therefore, to subdivide each of the major groups.

The Gradual High-Tone Loss Cases

On the basis of the lesions in the temporal bones the 46 ears with this type of hearing loss fall into four groups:

(A) The nerve atrophy is more severe than in the control group, but the middle-ear lesions are within normal limits when compared with those in the control group.

(B) The nerve atrophy and the middle-ear lesions are both more severe than are those in the control group.

(C) The nerve changes are within normal limits when compared with those in the control group, but the middle-ear changes are more severe than in the controls.

(D) The nerve and middle-ear changes are both within normal limits when compared with the control group.

Group A

The pathological findings typical for this group of 24 ears are shown in the photomicrograph (fig. 20) from the level indicated on the spiral graph of this ear (fig. 21). The audiogram for this ear is shown in fig. 22. The only lesion in the temporal bone which can account for the markedly lowered acuity for the higher tones in this patient is a partial atrophy of the nerve that involves most of the basal turn and is more advanced in the lower part than in the upper.

The data for the entire group are presented in linear graph form in table 2. They are arranged in the order of the thresholds for c^5 (4096 d.v.). This tone has been selected as the basis for arrangement because: (1) it is the intermediate of the three higher tones for which all

patients were tested; and (2) this tone (4096 d.v.) was heard by all of the ears in this group, whereas 8192 d.v. was not heard by 12 at the maximal intensities produced by the audiometers used.

Twenty of the 24 ears in this group are from individuals 59 years of age or older. The four ears from younger patients are all among those with the least nerve atrophy. The sex distribution is about even; ten are from females, fourteen from males.

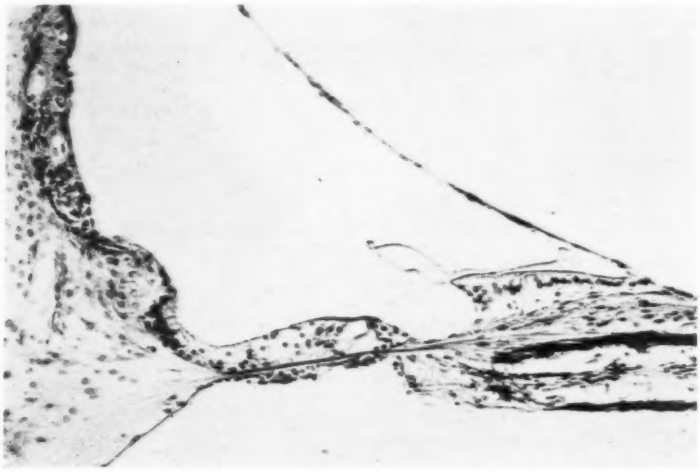


FIG. 20. Photomicrograph showing the typical lesion of the "gradual high-tone loss" group—a partial nerve atrophy in the basal turn. The organ of Corti, external sulcus cells and stria vascularis are normal. (This is from ear no. 12, table 2.)

The Middle Ear. While in no individual case are the middle-ear lesions more severe than are sometimes seen in the sections of ears with good hearing (the controls), yet there is a very high incidence of these minor lesions in the group as a whole. We call attention to this as a fact which should not be completely disregarded, even though we are unable to correlate these lesions with either the hearing loss or the atrophic changes in the cochlear nerve.

The Nerve. The nerve atrophy in these 24 cochleae is more ad-

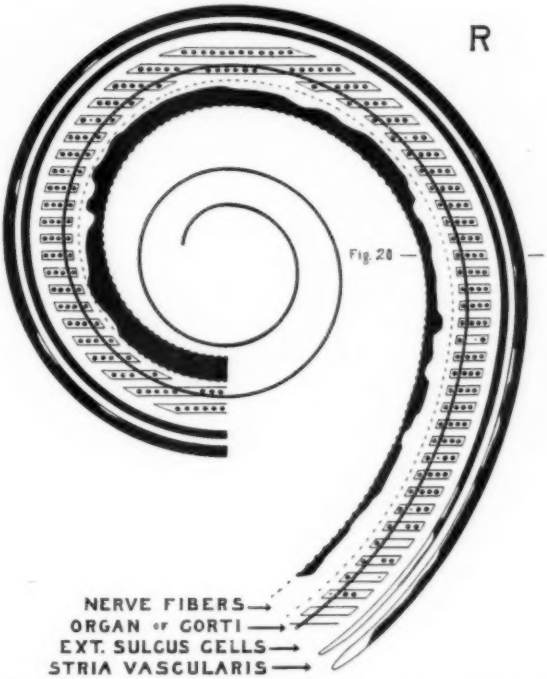


FIG. 21. The spiral graph of a cochlea with a "gradual high-tone loss" type of audiogram. (See also figs. 8 and 13.) The level of the section illustrated in fig. 20 is indicated.

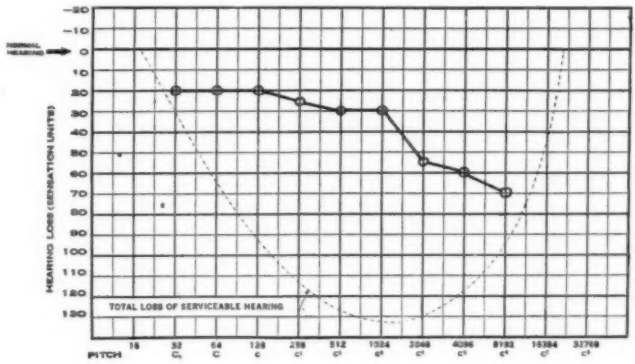


FIG. 22. Audiogram of the ear whose spiral graph is shown in fig. 21

vanced and more extensive than in any of the control group, but it is never total except at the extreme basal end. The atrophy extends beyond the 10 mm. level (*i.e.*, 10 mm. from the basal end), in all but one of the ears of the lower half of table 2, while there is marked involvement of this region in only one case in the upper half of the table. It is apparent that, in general, a relationship exists between extent of nerve atrophy in the basal turn and degree of high-tone loss.

It seems to be a fact that when the other structures in the middle and inner ear are within normal limits the threshold of acuity depends on the number of nerve fibers supplying the end-organ. This is illustrated by the fact that, for all but one of the 12 ears which did not hear 8192 d.v. at the maximum intensity of the audiometer, the upper limit for air conduction, as determined with the intense note of the Struycken monochord, was definitely higher than 8192 d.v. For seven ears the upper limit was either 12,000 or 13,000 d.v. This point, that the threshold of acuity depends on the relative number of nerve fibers supplying the end organ, is also well illustrated by a case reported in 1931 from this laboratory (5). In this patient, aged 63 years, the density of innervation of the organ of Corti at all levels in the better ear was only about one-fifth normal, as determined by ganglion cell counts. All tones from 64 to 8192 d.v. were heard, but only at intensities far above normal.

The Organ of Corti. It is characteristic of this group with a gradual high-tone loss that lesions of the organ of Corti are limited to the lower 2 or 3 mm. of the basal turn. This extent of lesion is but little greater than in some ears of the control group (see, for example, the spiral graph in fig. 17). Therefore, histologic changes of the organ of Corti limited to this part of the basal turn cannot explain the loss for the high tones under consideration, *i.e.*, up to and including 8192 d.v.

In a few ears there are at higher levels isolated sections with one or two hair cells missing, as indicated in table 2 by the vertical lines extending only one-fourth the width of the spaces. In one ear (no. 8) there is a definite end-organ lesion at the 8 mm. level, and as indicated in the table, marked atrophy of the external sulcus cells also is found at the same level.

The External Sulcus Cells. In only 3 ears of this group does the

atrophy of the external sulcus cells exceed that found in some of the controls. This is in marked contrast to the "abrupt high-tone loss" group (table 5) in which the frequent occurrence of severe lesions of the external sulcus cells suggests a possible functional significance (see p. 362).

The Stria Vascularis. The total amount of stria vascularis atrophy is in 22 ears of this group no greater than in the controls, and therefore cannot be regarded as a cause of the impaired hearing. Extensive and severe atrophy of the stria is found in both ears[4] (nos. 5 and 10, table 2) of a colored woman, aged 40 years. In this patient the amount of normal stria present is much less than in any of the other ears used in this study. The ear with the greater hearing loss (see audiograms, fig. 23) also has the more extensive strial atrophy. Photomicrographs of two sections from the ear with the worse hearing are shown in figs. 24 and 25, and their location is indicated on the spiral graph of this ear (fig. 26). Since the nerve atrophy is less than in other ears with a similar hearing loss and the external sulcus cells are normal at all levels in these two ears, it seems probable that the unusually extensive strial atrophy was an important contributing factor in the deafness of this patient.

The Basilar Membrane. When discussing the control group it was shown that hyalinization or the deposit of calcium salts in the lower 2 mm. of the basilar membrane causes no impairment of hearing for tones up to and including 8192 d.v. Changes involving more than 3 mm. of the basilar membrane are found in only 6 of the 24 cochleae in table 2. Mayer (8) has suggested that the impaired hearing of old age is due to increased rigidity of the basilar membrane. Five of these 6 ears are from the oldest patients in the group, but in all six the atrophy of the cochlear nerve, when compared with that in other ears without similar basilar membrane changes, fully accounts for the hearing loss.

[4] This is also the only case in which the saccular otolithic membrane is abnormal in appearance. In both ears it is greatly swollen and folded. The maculae and their nerves are normal. In view of the fact that the endolymph from the cochlea passes through the sacculus to reach the endolymphatic duct, the extensive atrophy of the stria may have been a factor in the causation of this "lesion" of the otolithic membrane.

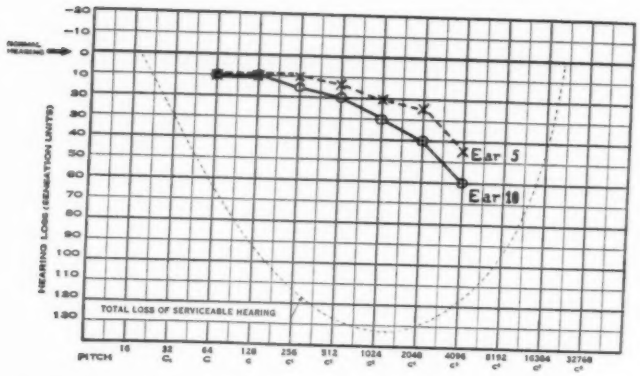


FIG. 23. Audiograms showing typical "gradual" high-tone losses in a patient (ears no. 5 and 10, table 2) with extensive atrophy of the stria vascularis (figs. 24, 25, 26).

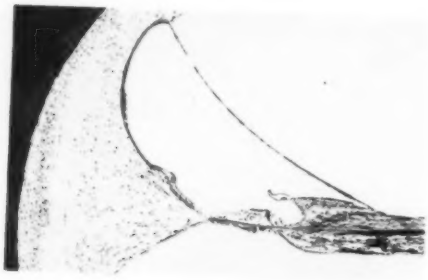


FIG. 24. Photomicrograph of section showing total atrophy of the stria vascularis and partial atrophy of the nerve in the lower basal turn of ear no. 10, table 2. Magnification, 58 diameters.

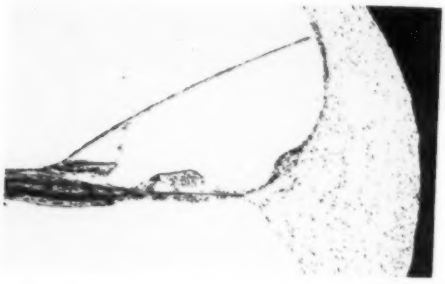


FIG. 25. From the area in the upper basal turn of ear 10, table 2, with total atrophy of the stria vascularis and normal nerve.

It is evident that in this "gradual high-tone loss" group of 24 ears the important lesion is partial atrophy of the nerve. The organ of Corti changes are of minor significance, in marked contrast to the important rôle they have in the "abrupt high tone loss" group (table 5).

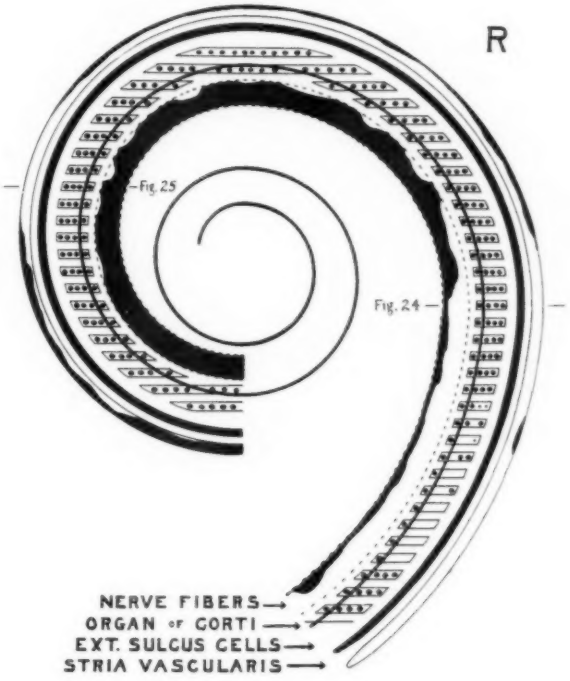


FIG. 26. The spiral reconstruction graph of ear 10, table 2. The audiogram is shown in fig. 23. The levels of the sections illustrated in figs. 24 and 25 are indicated.

A diagnosis of syphilis was made in only 3 of the 14 patients from whom these 24 ears were obtained (see table 2). It is evident from this fact that the cochlear lesions in these cases are not due to syphilis.

Autopsy revealed definite arterio- or arteriolosclerosis in all patients in this group (table 2). This is the only group of our entire material, however, in which any one systemic disease is found in all cases.

Even in the cases with the most extensive vascular sclerosis very little evidence is found of sclerosis of the labyrinthine artery or its branches within the cochlea.

The impaired hearing for high tones in the group of 24 ears which has just been considered is satisfactorily explained by the atrophy of

FIG. 27. Section of ear 25, table 3, showing a dense fibrous adhesion between the inferior surface of the anterior crus of the stapes and the promontory. It also shows a healed osteitis near the oval-window margin. Magnif., 36 diameters.

the cochlear nerve. Of the remaining 22 ears with a gradual high-tone loss those with severe middle-ear lesions have been carefully studied in an effort to determine whether or not middle-ear lesions can cause an impairment for high tones without affecting low-tone acuity.

Group B

The middle-ear lesions in the three ears (nos. 25, 26 and 27, table 3) of this small group are more severe than any in either the preceding

group or the control group. A partial atrophy of the cochlear nerve is also found in all three ears.

Two of these three ears (nos. 25 and 26) are from a 64-year old man who died from coronary artery occlusion two days after the hearing

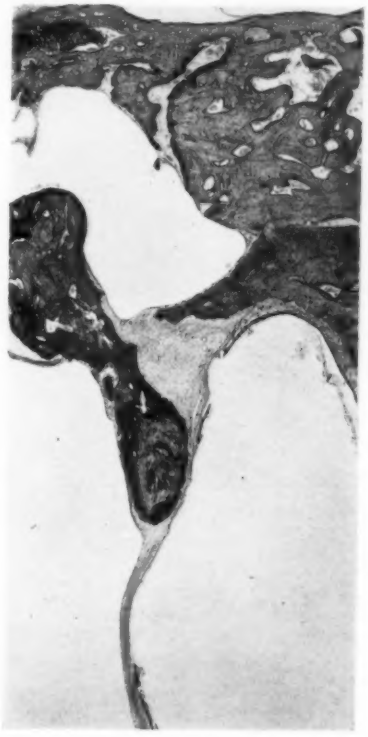


FIG. 28. The space between Shrapnell's membrane and the neck of the malleus is filled with dense fibrous tissue. (Ear no. 25, table 3.) Magnif., 10 diam.

examination. Air conduction time for a 512 d.v. fork was slightly shortened, bone conduction was not prolonged, and air conduction was much better than bone conduction; from the vertex lateralization was to the right. There is a history of ear-ache with discharge about 35 years previously. Otoscopic examination showed evidence of

marked middle-ear pathological changes. The more severe bilateral lesions seen in the sections are: (1) a dense fibrous adhesion of the inferior aspect of the anterior crus of the stapes to the promontory (fig. 27), and (2) the space between Shrapnell's membrane and the neck of the malleus is filled with dense fibrous tissue (fig. 28). The condition of the pars tensa of the tympanic membrane differs in the two ears; in the left there is marked fibrous thickening of the anterior

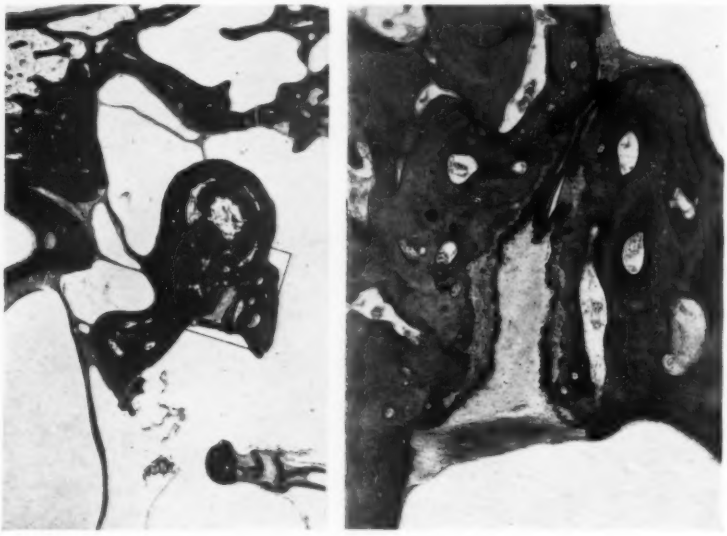


FIG. 29. Shows the partial ankylosis of the malleo-incudal articulation in ear no. 27, table 3. Magnifications, 7 and 33 diameters.

half and a slight degree of irregular thickening elsewhere; in the right there is a large atrophic scar in the anterior and inferior parts, with a marked degree of marginal thickening.

The third ear[5] (no. 27) also has a dense fibrous adhesion between the anterior crus of the stapes and the promontory. The rest of this middle ear is within normal limits except for a partial ankylosis of the malleo-incudal articulation (fig. 29). Air conduction for a 512 d.v.

[5] The opposite ear of this patient is no. 17, table 2.

fork was better than bone conduction, and bone conduction was not shortened.

From these three cases alone no conclusions can be reached as to the effect of these middle-ear lesions on high-tone perception, because in these ears the partial nerve atrophy in itself is sufficient to explain the hearing impairment.

Group C

The two ears (nos. 28 and 29, table 3) in this small group were obtained from a patient with a spinal-cord lesion who died suddenly

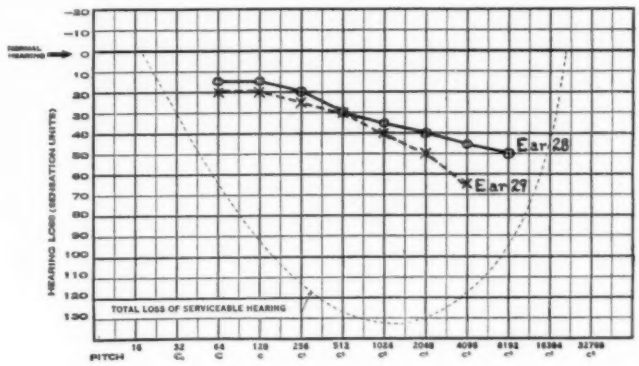


FIG. 30. Audiograms of a patient (ears 28 and 29, table 3). The hearing shown is typical of the "gradual" high-tone loss group; the lesions found are shown in figs. 31 to 34.

about 24 hours after the hearing tests were made. The most prominent lesions are in the middle ears, but both audiograms (fig. 30) are typical of the gradual high-tone loss cases and show that low tones were well heard and high tones poorly heard. Bone conduction time for a 512 d.v. fork was not shortened, and air conduction time was about 50 per cent longer than bone conduction. The 128 d.v. fork was lateralized to the left—the poorer ear for air conduction.

Otoscopic examination revealed marked pathological changes in the middle ear, and the histologic sections show (1) almost complete

destruction of the pars tensa of the tympanic membrane on the right (fig. 31) and a large atrophic scar on the left (fig. 32); (2) the space between Shrapnell's membrane and the neck of the malleus on both sides filled with cystic fibrous tissue; and (3) on the left the head of

FIG. 31. This section of ear no. 28, table 3, shows an almost complete destruction of the tympanic membrane, and Prussak's space filled with fibrous tissue. Magnif., 7 diameters.

the malleus embedded in myxomatous tissue in the epitympanic space (fig. 32). In both ears the oval and round window regions are essentially normal (fig. 33).

In contrast to the preceding group, the nerve atrophy in these two ears is within the range of the control group and cannot account for

the impaired hearing for high tones. Judging from the histologic appearance alone, the organ of Corti cannot account for the impaired hearing. The evidence presented on page 362, however, suggests a nutritional relationship between external sulcus cells and organ of

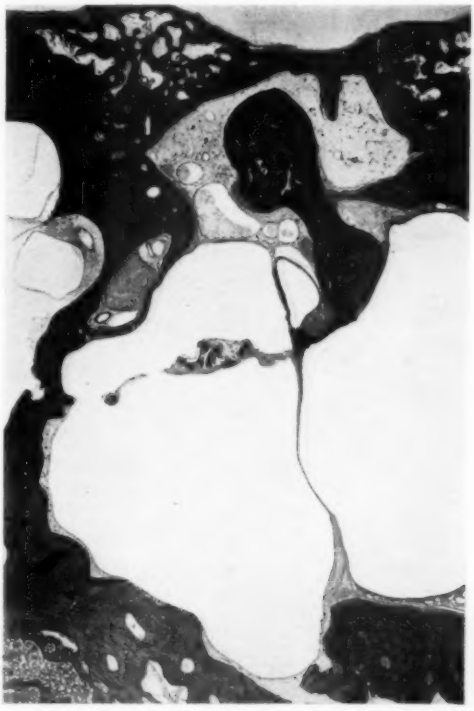


FIG. 32. This section of ear no. 29, table 3, shows the head of the malleus embedded in myxomatous tissue, Prussak's space filled with fibrous tissue, and an atrophic area of the tympanic membrane.

Corti; and it must be kept in mind that the atrophy of the external sulcus cells, which in this case is more extensive (especially in the ear with poorer hearing) than in any of the controls, may have been a factor in the causation of the high tone loss. In addition, an abnormality of Reissner's membrane must also be taken into account as a

possible cause of the hearing loss. At several levels in both ears the mid-portion of this membrane is thicker than usual; and in the left ear, in the region from 5.5 to 7 mm. from the basal end, it is much convoluted and consists of a layer of cuboidal cells on the ductus coch-

FIG. 33. (Ear no. 28, table 3.) The oval and round-window regions are normal. They are also normal in opposite ear (no. 29) of this patient.

learis surface with small blood vessels on the scala vestibuli side (fig. 34). Histologically, this resembles the choroid plexus. The tegmentum vasculosum of the bird ear, which corresponds to Reissner's membrane of mammals, has a somewhat similar appearance. This abnormality is unique in the entire collection of temporal-bone sec-

tions in this laboratory; therefore no conclusion can be drawn as to its effect upon the hearing of this particular patient.

The histologic findings in these two ears, obtained only one day after reliable functional tests, suggest that middle-ear lesions may cause marked impairment of hearing for high tones without noticeably affecting acuity for low tones. This is contrary to the usually accepted ideas, and the presence of other lesions in these ears makes it impossible to arrive at any definite conclusion.

FIG. 34. Section of ear no. 29, table 3, from the region in the upper part of the lower basal turn that has a much convoluted Reissner's membrane. Magnif., 58 diameters.

Group D

The 17 ears with gradual high-tone loss for which the data are given in table 4 have been grouped together because in none of them do the nerve, the organ of Corti or the middle-ear lesions afford an adequate explanation for the hearing impairment. In 11 of these ears no lesions have been found that differ from the lesions in some ears with good hearing for all tones up to and including 8192 d.v.

Four ears (nos. 34, 35, 44 and 46) have basilar membrane changes, of the type described by Mayer, more extensive than in the controls. In two of these ears with basilar membrane changes (nos. 35 and 44) and also in two other ears of this group (nos. 33 and 37) there is, in the

lower basal part of the scala vestibuli, a fibrous trabecula adherent to Reissner's membrane. Ear 44 also has total atrophy of the stria vascularis between the 6 and the 9 mm. levels, and in addition an unusual amount of pigment in this area. Free clumps of pigment granules are seen in the lumina of the lower basal turn of the ductus cochlearis, the canalis reuniens and the sacculus. The metabolism of the end-organ of the basal turn may have been unfavorably affected by this strial abnormality, and of the three lesions in this ear we regard it as offering the best explanation for the impaired acuity for high tones. The fibrous trabeculae in the scala vestibuli are probably due to incomplete resorption of mesenchyme during development; they have not been found in any ears of the control group or of groups A, B or C, but we do not believe that they can have much effect upon the hearing. A definite conclusion with respect to the effect of basilar-membrane changes upon hearing is precluded by the fact that no lesion observed in the temporal-bone sections offers an explanation for a similar impairment of hearing in the remaining 11 ears of table 4.

When all 46 ears with gradual high-tone losses are listed in the order of acuity for 4096 d.v., 15 of the 17 ears of group D fall in the upper half of the list. Of the several hypothetical explanations that might be advanced for the impaired hearing for high tones by these ears in group D, which do not have the lesions usually found in those ears with more marked high-tone impairments, only two will be mentioned. (1) There may be an atrophy of part of the non-medullated collateral and terminal nerve fibers within the organ of Corti itself. We have been unable to demonstrate enough of this elaborate plexus to justify an opinion as to its condition relative to the normal. (2) There may be minute lesions of the auditory pathways or centers of the central nervous system that can only be demonstrated histologically. As stated previously, the funds of the laboratory are not sufficient to permit the preparation of serial sections of the central nervous system. We can state definitely, however, that gross intracranial lesions do not account for the impaired hearing by any of the ears used in this study.

The Abrupt High-Tone Loss Cases

This group of 33 ears differs very decidedly from the preceding group: the loss of acuity is "abrupt" instead of a gradually increasing

impairment for each succeeding high tone; the extent of end-organ and external-sulcus-cell lesions is usually much greater than in the "gradual" loss group (table 2), and total atrophy of the nerve to regions of the basal turn is frequently observed. These are the main differences between the two big groups so far as the audiogram and the pathology are concerned. From the point of view of interpretation of clinical tests of hearing it is encouraging that about 75 per cent of the 33 ears with the "abrupt" type of impairment for high tones have, in varying degrees, an atrophy of the organ of Corti, of external sulcus cells and of nerve in the same general location (the lower basal coil).

Although these 33 ears have in common an "abrupt" impairment for one or more of the high tones, the pathology is not uniform in all. On the basis of the histological lesions in the temporal bones they fall into four groups:

(E) In 24 ears the most prominent lesion is atrophy of the organ of Corti and nerve in the basal coil. The middle-ear lesions are within normal limits when compared with those in the control group.

(F) In 3 ears the organ of Corti is normal in appearance, but the nerve atrophy in the basal coil is far greater than in the controls. The middle-ear lesions are within the range of the controls.

(G) In 2 ears the organ of Corti is normal in appearance, but the nerve atrophy and the middle-ear lesions are both more severe than in the controls.

(H) In 4 ears the organ of Corti, nerve and middle-ear changes are all within normal limits when compared with the control group.

Group E

The range of variation in the audiograms of this group of 24 ears is shown in fig. 35. Among the several thousand clinical records of hearing in the laboratory are many audiograms with an "abrupt" drop in acuity at 512 or 1024 d.v., but we have as yet no histologic sections of an ear with this very severe type of hearing loss.

The atrophy of the organ of Corti characteristic of this group may be continuous throughout the entire lower basal turn, as in fig. 36, or limited to an area less than one millimeter in length (fig. 37). The histologic lesions typical of this group are shown in figs. 38 to 40,

from the levels indicated on the first spiral graph, and in figs. 4, 5, 6 and 10.

The cochlear lesions and other data for these 24 ears are recorded in table 5, and comparison with those in table 2 brings out better than any written narrative the essential difference in the pathology of the "abrupt" and the "gradual" high-tone loss groups. The ears in table 5 are not arranged in the order of their acuity for the tone 4096 d.v., as in the previous tables, but according to the number of high tones involved. Thus 2 of fig. 35 is placed below 1, and 3 is below 2.

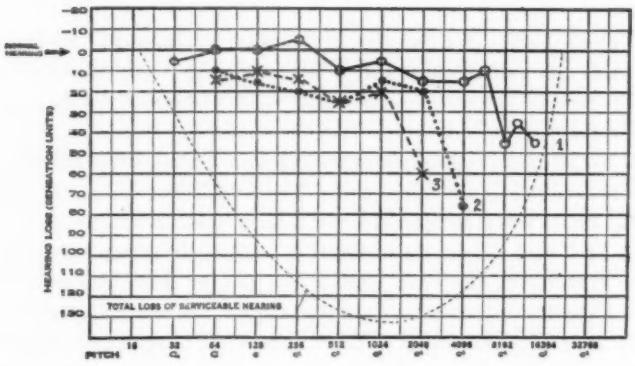


FIG. 35. These three audiograms show the range of variation in the audiograms of group E. Audiograms 1, 2 and 3 are, respectively, those for ears 47, 53 and 69 of table 5.

The Middle Ear. Middle-ear lesions more severe than in the controls are not found in any ears of this group.

The Nerve. Total atrophy of the nerve is extensive in some cases, in others it is localized. There is always total atrophy of the organ of Corti in the regions in which the nerve has entirely disappeared (see figs. 6 and 10). On the other hand, in some sections with total absence of the organ of Corti a few nerve fibers are seen in the osseous spiral lamina near the foramina nervosa. Contrary to the reports of Manasse (9) and others, we have not seen, in these cases with an advanced or with any lesser degree of nerve atrophy, a definite increase in fibrous tissue or any evidence of an inflammatory reaction in

the nerve canals of the osseous spiral lamina. Only a few loose strands of tissue are seen in the spaces normally filled with nerve. Furthermore, we have not seen nerve fibers in the process of degeneration. The histologic technique we use is not suitable for the detailed study of degenerating nerve fibers, but if such a process was going on, some evidence of it should be seen in our sections.

The transition from total absence of nerve fibers to normal nerve within the space of one millimeter or less is often seen in this group in which there is an "abrupt" and marked functional loss of hearing for tones an octave apart. These abrupt changes in the nerve are not found in any sections of the "gradual" high-tone loss group.

The Organ of Corti. The most distinctive histological lesion in this group of ears with an abrupt high-tone loss is atrophy of the organ of Corti at some level above the last 2 mm. of the basal turn. The atrophy of the organ of Corti, however, is always limited to the basal turn. The area of atrophy may be continuous for as much as 10 mm., or it may be limited to a region less than one millimeter in length (figs. 36 and 37). The extensive and continuous type of atrophy is present in 10 ears, and the more localized or "patchy" type in the remaining 14 in this group. Ears nos. 65 and 70 (table 5) are from a patient with an extensive atrophy in one ear and areas of localized atrophy in the other. In ear no. 50, both the impaired hearing and the atrophy of the end-organ are on the left; the right ear is normal.

The extent of the atrophic area of the organ of Corti is not related to age. The ages of the 4 patients with the most extensive atrophy were between 38 and 46 years. There is no evidence that a damaged organ of Corti regenerates, so it must be due to chance alone that so many with this type of lesion are among the younger patients. Further study of this point may lead to a suggestion of the etiology.

All of the 24 ears in this group are from males. Bunch and Raiford (10), in 1931, observed that when the audiograms of males and females are separately grouped by decades the average acuity of hearing for the higher frequencies is always better in the females. Ciocco (11), with a different method of analysis and a larger number of cases, found a similar sex difference in the acuity for high tones. We have found extensive atrophy of the organ of Corti in but one female. The lesion is certainly more common in the males.

The External Sulcus Cells. Table 5 shows that, whether the total atrophy of the organ of Corti be extensive or localized, the external sulcus cells are always atrophic in the same area. This suggests a nutritional relationship between these two structures. Often the

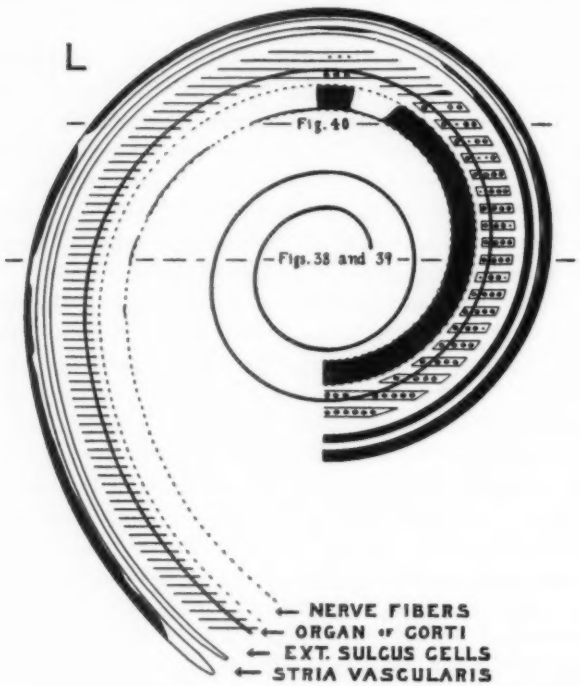


FIG. 36. The spiral reconstruction graph of the cochlea of an ear (no. 70, table 5) with an "abrupt" high-tone loss and extensive atrophy of the organ of Corti, nerve and external sulcus cells in the basal turn. The levels of the sections illustrated in figs. 38, 39 and 40 are indicated.

extent of external-sulcus-cell atrophy is greater than that of the end-organ atrophy. It is our impression that the changes in the external sulcus cells, as seen in the sections, precede the end-organ lesion, and that the effect on the organ of Corti of a local atrophy of these cells is not compensated by an increased activity at other levels.

The Stria Vascularis. In 19 of these 24 cochleae the atrophy of the stria vascularis is no greater than is seen in some of the control group, so it seems unlikely that the lesions of the stria in these ears are in any way responsible for the atrophy of the organ of Corti or for the

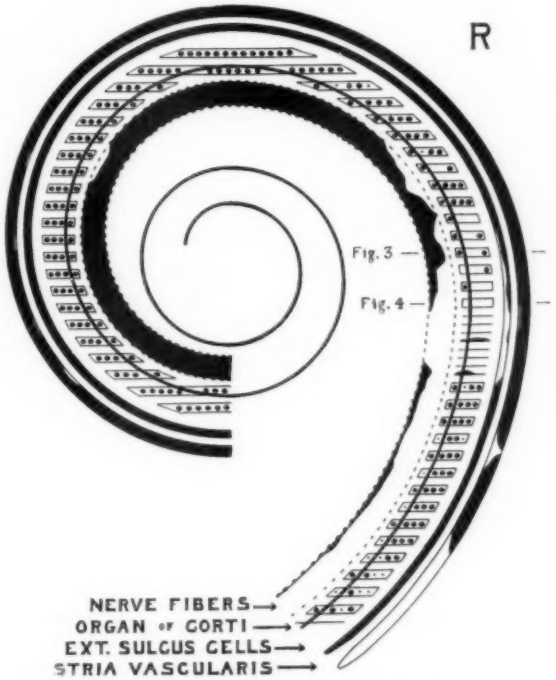


FIG. 37. The spiral reconstruction graph of the cochlea of an ear (no. 48, table 5) with an "abrupt" high-tone loss but only a limited area of organ of Corti atrophy. The levels of the sections illustrated in figs. 3 and 4 are indicated. (The section 0.24 mm. basalward from that of fig. 4 shows total atrophy similar to that shown in fig. 6.)

hearing loss. In 5 ears the strial changes are more extensive than in the controls, but unlike what is seen in the external sulcus cells, the regional distribution of the lesions does not correspond with the areas of end-organ atrophy (see table 5). Also, the extent and location in

the basal coil of the organ of Corti and nerve atrophy fully account for the loss of high tones in these 5 ears. Therefore, in this group it is impossible to ascribe any part of the hearing loss to the atrophy of the stria vascularis. In a previous group (p. 347), the extensive bilateral atrophy of the stria vascularis in one patient seemed to be an important factor in the causation of the deafness.

The Basilar Membrane. The basilar membrane changes are within normal limits in 19 of the cochleae in this group. Definite calcifica-

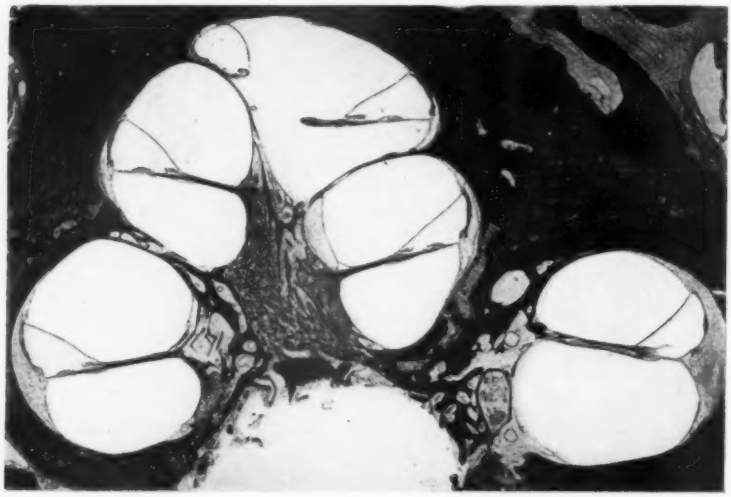


FIG. 38. Photomicrograph of section of ear no. 70, table 5, from level indicated in spiral graph (fig. 36). Magnif., 15 diameters. The structures seen in this section are normal in all turns but the lower basal.

tion or hyalinization are seen in this structure in 5 ears, but the effect of these lesions on the hearing cannot be determined because the organ of Corti and the nerve are also diseased.

Fibrous Trabeculae in the Cochlea. Fibrous trabeculae were found in the scala vestibuli of the lower basal turn in one ear in this abrupt high-tone loss group (no. 61, table 5). This abnormality was also found in 4 ears in the gradual high-tone loss group (table 4). It is sug-

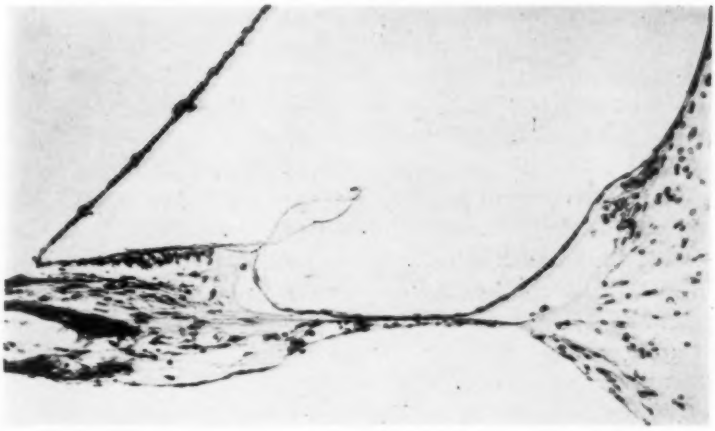


FIG. 39. The lower basal turn of the section shown in the preceding figure, at a magnification of 150 diameters. This shows the typical lesion of "abrupt" high-tone loss—total atrophy of organ of Corti and external sulcus cells, and a severe degree of nerve atrophy. The tectorial membrane is, for this method of histologic preparation, always normal in appearance in such areas. (The "lifting" of the cells of both external and internal sulcus regions is an artefact.)

FIG. 40. Photomicrograph of section of ear no. 70, table 5, from level shown in spiral graph (fig. 36). There is total atrophy of the organ of Corti and external sulcus cells in the lower basal part of this section (to the right in the figure); and these structures are normal in the upper basal part. Near the middle of the osseous spiral lamina can be seen the short region of normal nerve between areas of marked atrophy, as shown in the spiral graph. Magnif., 15 diam.

gestive that the hearing loss in the 5 cochleae with this abnormality is worse than would be expected from the lesions in other structures.

Comparison of tables 2 and 5, with respect to the cochlear lesions and the information from the clinical histories and pathological records regarding systemic vascular disease, shows that the more severe cochlear lesions are in the group with the least vascular pathological change. The average of the blood pressures is considerably lower and the histologic evidence of advanced arteriosclerosis is decidedly less in table 5 than in table 2. The cochlear lesions in table 5 are much more extensive, although the average age of these patients is 15 years less than in table 2. These facts show that there is no relation between systemic vascular disease and severity of cochlear lesions.

Only one patient in table 5 and three in table 2 had syphilis. Histologically, their ears are so similar to those of the non-syphilitic patients in the respective groups that there is no reason to regard syphilis as the cause of the lesions which account for their impaired hearing.

The remaining nine ears of the patients with abrupt high-tone loss are divided between groups F, G and H. The data for these three small groups are given in table 6. All nine of these ears are "exceptions" in that the organ of Corti is either normal or the atrophy does not exceed that seen in some cochleae with good hearing for all tones up to 8192 d.v. When all 33 ears with abrupt high-tone loss are arranged in the order of severity of hearing impairment, these nine "exceptions" fall in the upper half of the list.

Group F

The hearing for the first three ears in table 6 is of the abrupt high-tone loss type, but the cochlear lesions are typical of those shown in table 2 for the gradual high-tone loss group; *i.e.*, a partial atrophy of the cochlear nerve is the most prominent lesion. The spiral reconstruction charts of ear no. 72 of table 6 and ear no. 22 of table 2 are very similar (figs. 41 and 42), but the audiograms (fig. 43) differ in that one is typical of the abrupt and the other of the gradual high-tone loss groups. We can offer no explanation for the fact that these

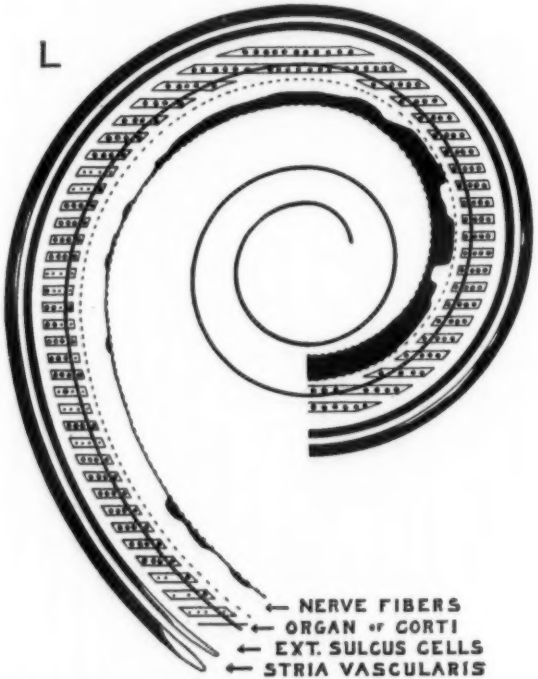


FIG. 41. The spiral reconstruction graph of ear no. 72, table 6

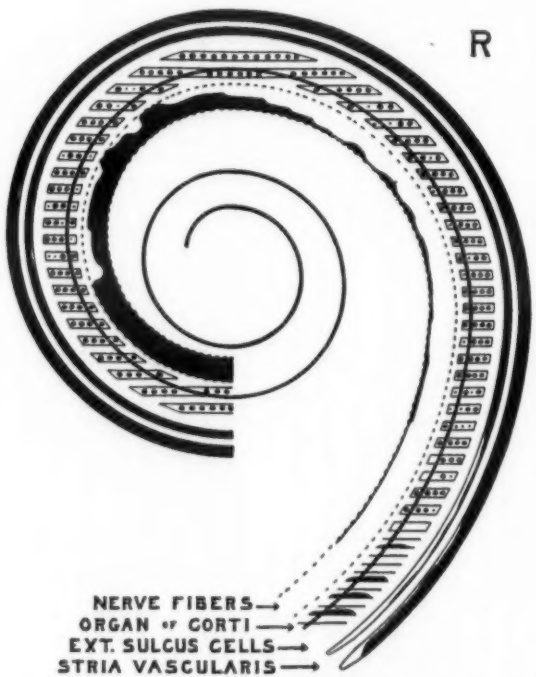


FIG. 42. The spiral reconstruction graph of ear no. 22, table 2

three ears do not have the type of hearing loss usually associated with a partial atrophy of the nerve and an intact end-organ.

Group G

These two ears (nos. 74 and 75, table 6) are similar to the three ears of group B (table 3, ears 25, 26 and 27) with a gradual high-tone loss, in that the organ of Corti is normal and the nerve atrophy and middle-ear lesions are both more severe than in the controls. For the study of the effect of middle-ear lesions upon hearing neither of these

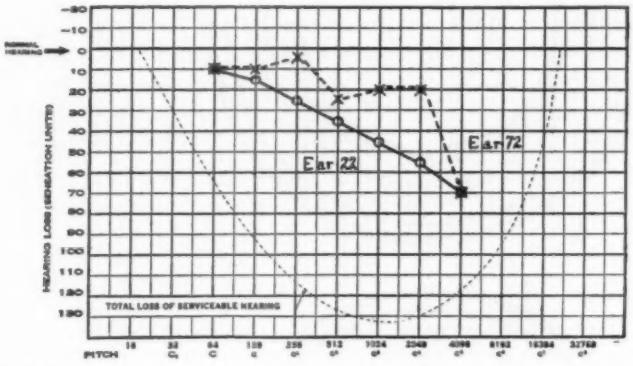


FIG. 43. The audiograms of the ears whose spiral reconstruction graphs are shown in figs. 41 and 42. The cochlear lesions in both are very similar; they account for a high-tone loss but not for the difference in type of impairment.

two ears is of much value, because (1) for ear 74 no tuning-fork tests were made owing to extraneous circumstances, and (2) in the removal of ear 75 at autopsy the middle ear was so broken that the upper half of the tympanic membrane, the epitympanic region and the malleus and incus are missing in the sections. In both ears there is a fibrous adhesion between the anterior crus of the stapes and the promontory, but not so extensive as in the ears of group B. In ear 74 Shrapnell's membrane is retracted until adherent to the neck of the malleus, and the space between the head of the malleus and the lateral wall of the attic is filled with myxomatous tissue.

The nerve atrophy alone in each of these two ears is sufficient to

account for an impairment of acuity for high tones; in fact, the hearing for ear 75, tested 12 days before death, was much better than is usual for the amount of nerve atrophy. The anatomical differences with respect to the nerve atrophy and the middle-ear lesions in the five ears of groups B and G offer no satisfactory explanation for the fact that three have a "gradual" and two an "abrupt" type of high-tone loss.

Group H

The last four ears in table 6 have no lesions in the middle ear, nerve, organ of Corti, external sulcus cells, stria vascularis or basilar membrane that are more severe than in some of the control group. Two of these ears (nos. 77 and 78) are from a man with advanced cerebral arteriosclerosis. In ear 79 there is a fibrous trabecula adherent to Reissner's membrane in the scala vestibuli of the basal turn. The other ear (no. 76) has the least hearing impairment of any of the 33 ears with abrupt high-tone loss. As with group D, we do not believe that the lesions found by histologic examination of these four temporal bones caused the hearing impairments observed.

LOCALIZATION

Some of the cases of abrupt high-tone loss are particularly suitable for the study of tone localization in the human cochlea. The 8 ears of group E (table 5) which have the most extensive and continuous areas of organ of Corti atrophy, furnish the best evidence as to the localization of certain high tones. In fact each of these cases is an experiment from the viewpoint of the physiology of the inner ear.

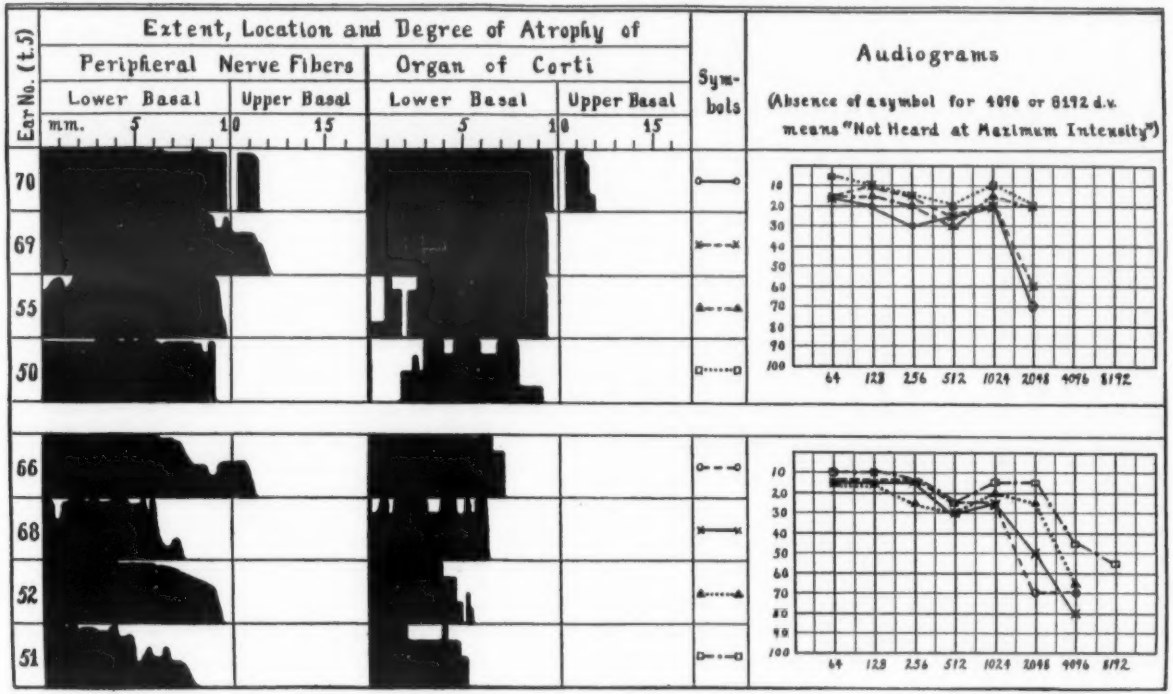
The evidence from two ears,[6] included in a publication (5) from this laboratory in 1931, indicated that (1) the lower basal turn of the cochlea is the part most concerned in the hearing of tones higher than 3000 d.v., and (2) the area for 4096 d.v. is about 8 to 9 mm. and that for 8192 d.v. is about 5 mm. from the basal end of the cochlea. The

[6] One of these ears had a marked impairment for 4096 d.v. but good hearing for the tones only a half octave to either side and for all other high tones up to and including 10,321 d.v. The other ear had an abrupt high-tone loss and is therefore also included in this paper (no. 52, tables 5 and 7).

TABLE 7

The data as to the extent and location of the lesions of the organ of Corti and the nerve, together with the respective audiograms, of the 8 ears of group E which provide direct evidence as to cochlear localization of response to certain tones

(The serial numbers used are the same as in table 5, to which reference may be made for the other data regarding these ears)



correlations of hearing and cochlear lesions in this larger group (table 7) confirm the earlier conclusions.

The data regarding the lesions of the nerve and of the organ of Corti, and the audiograms of these 8 ears, have been arranged in this table in such manner as to facilitate comparisons. The order of arrangement is based on the extent of atrophy of the organ of Corti, which varies from 12 mm. in the first case to about 5 mm. in the last two ears. Atrophy of the nerve is in every case at least as extensive as the end-organ atrophy and in three it is more extensive. As a group these 8 ears furnish strong evidence in favor of some form of localization of cochlear response to sound stimuli, since there is a positive correlation between the extent of the atrophic area above the basal end of the cochlea and the number of high tones affected.

The tone of c^3 (1024 d.v.) was heard within normal limits by the ears with the most extensive atrophy. Hence we can state definitely that the area of response to stimulation by this tone (1024 d.v.) is more than 12 mm. from the basal end of the cochlea. This must also be true for all tones of lower frequencies than 1024 d.v. per second.

The tone of c^4 (2048 d.v.) was the highest one heard by any of the first four ears in table 7. Ear no. 70 has the greatest impairment of hearing for this tone and also the greatest extent of end-organ atrophy. The audiogram shows that the threshold acuity for 2048 d.v. was greatly impaired (70 decibels). With the exception of a small area near the 10 mm. level there is atrophy of both nerve and organ of Corti for 12 mm. from the basal end. The tones used in making the audiometer tests were at octave intervals and we have no information as to the functional significance of this circumscribed area with normal-looking nerve and end-organ. This tone (2048 d.v.) was also very poorly heard by ear 69, in which there is total atrophy of nerve and organ of Corti for 9.5 mm. from the basal end, and a marked degree of partial nerve atrophy up to the 11.5 mm. level. The other two ears (nos. 50 and 55) heard this tone within normal limits (20 decibels), and in these ears the upper end of the area of atrophy of both nerve and organ of Corti is near the 9.5 mm. level. Both ears have some normal-looking organ of Corti near the basal end of the cochlea, but there are only a few nerve fibers supplying these regions. Since the upper end of the area of organ of Corti atrophy is at the same

level in ears 55 and 69, the difference of 40 decibels in the threshold for 2048 d.v. can only be explained by the partial nerve atrophy between the 9.5 and 12 mm. levels in ear 69. These cases indicate that the lower end of the area of response to stimulation by 2048 d.v. is more than 9.5 and less than 12 mm. from the basal end of the cochlea.

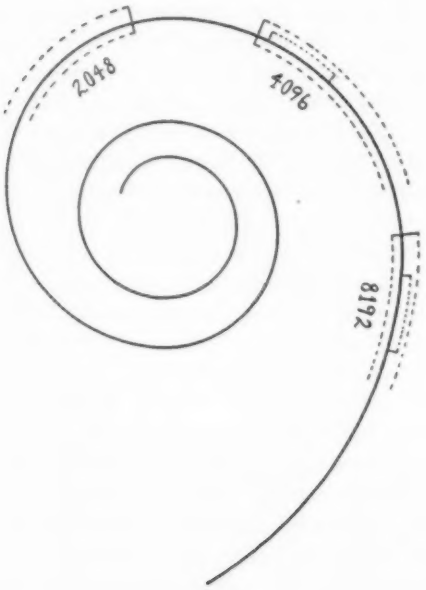


FIG. 44. Spiral diagram of the cochlea showing the localization indicated by our material. The smaller areas marked out for 4096 and 8192 d.v. are those indicated by Ciocco's statistical analysis of the spiral graphs.

Our data do not establish even an approximate upper boundary to the area for this tone.

The tone of c^5 (4096 d.v.) was not heard by any of the first four ears of table 7, but was heard by all of the second group of four ears. For three of these ears it was the highest tone heard. Of the ears which did hear this tone the one with the most extensive atrophy is no. 66, which has no hair cells in any part of the organ of Corti of the lower

7.3 mm. of the basal turn. Since this tone was not heard by the ears with organ of Corti atrophy to the 9.5 mm. level, the conclusion may be drawn that the upper boundary of the area that can respond to stimulation by 4096 d.v. is definitely more than 7.3 mm. and less than 9.5 mm. from the basal end of the cochlea. The lower boundary of the area of response for 4096 d.v. cannot be determined from our data, because this tone was not heard within normal limits by any of the 8 ears.

The tone of c^6 (8192 d.v.) was heard by only one ear (no. 51). In this ear the extent of the organ of Corti atrophy is about the same as in ear 52 (5 mm. from the basal end); there is partial nerve atrophy beyond this level in both ears but it is less marked in the one which did hear 8192 d.v. This indicates that the upper boundary of the area which can respond to stimulation by 8192 d.v. must be more than 5 mm. from the basal end of the cochlea. The lower boundary of the area for this tone cannot be determined from these cases.

The number of cases is not sufficient to justify any positive statement as to sharp localization, especially since the tones used in making the hearing tests were at octave intervals. As additional cases are studied in this way it may be possible to determine more definitely the area for the reception of these various tones (2048, 4096 and 8192 d.v.). The approximate areas for these tones, as determined from these 8 ears, are indicated graphically in figure 44. The lower boundary of the area for 2048 d.v. is shown as at the midpoint of the limits indicated by our cases; an upper boundary is not shown because this tone was heard by even the ear with the most atrophy. The upper boundaries of the areas for 4096 and 8192 d.v. are shown as at the 9 mm. and 5.5 mm. levels, respectively; lower boundaries for these areas are not shown because neither tone was well heard by any ear of the group.

The spiral reconstruction charts and the audiograms for all of our high tone loss cases, including the 8 cases just analyzed in detail, have been studied from the viewpoint of localization by Ciocco (12), who used a statistical method. The method consists of making a series of comparisons of the degree of nerve atrophy at all levels of the cochlea with the hearing impairment for each tone. For 4096 d.v. the best agreement between the amount of nerve and the hearing for this tone

is in the region 7⅛ to 8⅛ mm. from the basal end. For 8192 d.v. a similar agreement obtains for the area 4 to 5 mm. from the basal end (see fig. 44). The conclusions reached with these two methods as to the localization for 4096 and 8192 d.v. are in good agreement.

DISCUSSION AND CONCLUSIONS

An intelligent interpretation of audiograms and other hearing tests depends on our knowledge of the physiology and pathology of the ear. From this viewpoint pathological studies are most valuable when correlated with reliable hearing tests. For the past ten years we have been engaged in improving our methods of testing hearing and of preparing temporal bones for histologic study. Audiometers have proved to be more dependable than other methods of determining the hearing acuity, especially for air conduction and for the higher tones.

This publication is a correlation of the clinical and pathological findings in 79 ears whose audiograms show impairment of hearing limited to the high tones. A proper control for this study has been established by comparing the middle and inner ear lesions in ears with known good hearing for all tones with the lesions found in our cases with impaired hearing. The control group numbers about 200 ears.

Our observations prove very definitely that the receptors for high tones are located in the basal turn of the cochlea. Three-fourths of the ears with impaired hearing for high tones have lesions of the basal turn more extensive and severe than in any of the control group.

The audiogram is of the "abrupt" high tone loss type for all of the ears in which there is an area of atrophy of the organ of Corti more than 2 mm. from the basal end. The prominent lesion in the majority of the ears with the "gradual" type of high tone loss is a partial atrophy of the cochlear nerve supplying the basal turn. There are exceptions in that some ears with the "abrupt" type of loss do not have an atrophy of the organ of Corti; also some ears with the "gradual" type of loss do not have a sufficient degree of nerve atrophy to explain the impairment of hearing. In approximately one-fourth of the entire group of 79 ears no lesion was found in either the middle or inner ear that adequately explains the hearing loss. These ears have less hearing impairment than the group as a whole, and it is possible that

their hearing losses were due to early organic changes in the cochlea which our histologic technique is not yet good enough to demonstrate. On the other hand, the impaired hearing in these cases may be due to lesions of the central auditory pathways.

In regions in which the nerve has entirely disappeared from the distal part of the osseous spiral lamina, there is always total atrophy of the organ of Corti. In regions in which the nerve has not entirely disappeared, but a few normal-looking nerve fibers remain in the osseous spiral lamina, the organ of Corti may be either normal in appearance or atrophied. These facts indicate that nerve atrophy does not necessarily precede organ of Corti atrophy. This conclusion is strengthened by the fact that in our material the average age of the patients with extensive atrophy of the organ of Corti is 15 years less than that of the patients with a partial nerve atrophy as the most severe cochlear lesion.

Normal-looking blood vessels are present in our sections in regions with total atrophy of the organ of Corti. The fact that the areas of end-organ atrophy are often very sharply circumscribed suggests occlusion of terminal arteries as the cause; we have no histological evidence, however, to support this view. The clinical and pathological evidences of systemic vascular disease, as recorded in our tables, also indicate that arterio- or arteriolosclerosis is not the cause of organ of Corti atrophy. On the other hand, in patients with severe degrees of arterio- or arteriolosclerosis a partial atrophy of the nerve to the basal coil is commonly found, and the audiograms of most of these patients show a "gradual" rather than an "abrupt" type of high-tone impairment.

For only 9 of the 79 ears can syphilis be considered as a possible factor in the causation of either the cochlear lesions or the impaired hearing. The histologic lesions in these 9 ears differ in no way from those in the ears of patients without clinical or pathological evidence of syphilis. It is our impression that syphilis does not cause high-tone impairment without at the same time involving low-tone perception.

There is considerable evidence of a nutritional relationship between the external sulcus cells and the organ of Corti. The area of external sulcus cell atrophy is always at least equal in extent to that of the

organ of Corti atrophy, and frequently it is more extensive. In some cochleae with the organ of Corti normal in appearance external sulcus cell atrophy is found over a considerable area. It is our impression that the external sulcus cell atrophy precedes the end-organ atrophy, causing a functional impairment of the organ of Corti before definite histologic changes are recognizable. We have no information that even suggests an etiological factor for external sulcus cell atrophy.

An extreme degree of atrophy of the stria vascularis must be present before any effect upon the hearing can be demonstrated. This condition was found so infrequently in these ears that we conclude that high-tone impairment is seldom due to lesions of the stria vascularis.

Tinnitus is not a prominent symptom in high-tone impairment. It was recorded as present in 21 of the 79 ears; but in none was it the distressing symptom so commonly observed in patients with otosclerosis or other lesions of the conductive apparatus. No cochlear lesion has been found in the ears with a history of tinnitus that is not also present in ears without this symptom.

Study of the control group shows that certain middle-ear lesions are not infrequently found in ears with good hearing for all tones. Middle-ear lesions more severe than in any of the control group were found in only 7 of the 79 ears; in each of these 7 ears, however, the cochlear lesions are sufficient to explain the high-tone impairment. Air conduction was definitely better than bone conduction in all of the 79 ears in which it was possible to make tests with tuning-forks: this includes 6 of the 7 ears with middle-ear lesions.

It was possible to make caloric vestibular tests in 38 of these 79 ears. Normal reactions were obtained from all of the ears tested. In only 4 of the entire group of 79 ears were histologic lesions of any part of the vestibular apparatus found.

Our observations definitely prove that there is some form of localization of cochlear response to certain high tones, but do not favor any particular theory, such as that of Helmholtz or of Ewald.

Our histologic observations show that for the interpretation of clinical tests of hearing the frequency of 4096 d.v. is of particular importance. From the evidence presented for the ears with a "gradual" high-tone loss, a partial atrophy of the nerve supplying the basal turn of the cochlea is almost certain to be present when the

hearing impairment for 4096 d.v. is 60 decibels or more. There is much less certainty of a cochlear lesion when audiograms of this type show a loss of less than 50 decibels. The tone of c^5 (4096 d.v.) was heard by all of the 46 ears with the "gradual" type of high-tone loss, but the hearing for this tone was completely lost in 4 of the 33 ears of the "abrupt" high-tone loss cases. Histologic examination of these 4 ears shows an extent of organ of Corti atrophy greater than in any of the others. Therefore, an inability to hear 4096 d.v., when low tones are well heard, indicates an extensive lesion of the organ of Corti in the basal coil of the cochlea.

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EXPLANATIONS FOR TABLES 1 TO 6

General purpose of arrangement: to assemble all pertinent information concerning these ears into a form which facilitates the making of comparisons of both the groupings we have made and of the individual ears. For each group the average condition of each structure or group of structures—such as the middle ear or the blood vessels—can be seen at a glance, while the detailed tabulation enables the reader who may be especially interested in the effect on hearing of any particular part or condition to utilize our data for his own studies.

For the large section of the tables headed "*Histologic Observations of Conditions in the Basal Turns of the Cochleae*," see Method of Study, and especially figs. 2 to 6 and 11 to 15. This explains the symbols for *Nerve Fibers*, *Organ of Corti*, *Ext. Sulcus Cells* and *Stria Vascularis*. The first column under Basilar Membrane (*Bas. Memb.*) gives the extent of the area, in mm. from the basal end of the cochlea, with calcification (*Calcif.*) of the basilar membranes; and the second column (*Ca. + Hy.*) the total length of the area of basilar membrane with calcified and "hyalinization" appearances. (We use the term "Hyalinization" (*Hy.*) for the lesser degree of change, but recognize that this is not truly descriptive of the condition.)

The data for "*High-Tone Acuity*" are given in terms of the readings of the Western Electric Co. audiometers; the figures are for the decibels of increased intensity required to reach the threshold of hearing. For the conditions of our examinations readings from 0 to 20 are all within the range of variation of people with good hearing (Bunch and Raiford, 1931). Inability to hear a tone at the maximal intensity produced by the audiometer is recorded as *nh.* (not heard).

Monochord—AC: the figures give, in thousands of double vibrations, the upper limit for air conduction as determined with a Struycken monochord. For this column *nh.* means "not heard at the lowest frequency for longitudinal vibrations;" i.e., the upper limit is less than 55.0 d.v.

The other columns of the tables are considered below, in order from left to right, for such items as are not self-explanatory.

Ear Number: For convenience of reference all ears with high-tone impairment are given consecutive numbers, from 1 to 79, through tables 2 to 6. The ears of the control group (table 1) are not numbered serially with those having impaired hearing. The order of arrangement of ears in each table is explained in the text for the group.

Autopsy Number: The five digit numbers are those for the pathology service at the Johns Hopkins Hospital (these patients were tested with a 1-A audiometer); the four digit numbers are for the pathology service at the Bay View Division of the Baltimore City Hospitals (these patients were tested with a 2-A audiometer).

Race: W = white; *C* = colored (negro).

Because of the large amount of space it would require to give in even abbreviated words the observations of the otoscopic examinations and the histologic study of the middle-ear structures, the data for these items have been reduced

to a simple code form. The general principle of the code used is that *n* = normal, and *n* −, *1* and *2* record lesions within the range of those found in patients with good hearing for all tones; while the higher numbers (*3*, *4* or *5*) indicate the more severe lesions.

Otoscopic: Examples of the conditions recorded as *n* − are:—"slight to moderate retraction, otherwise normal;" "dull appearance, somewhat retracted;" etc. Examples for *1* are:—"moderate diffuse thickening;" "definite marginal thickening with slight diffuse thickening elsewhere;" "healed scar, otherwise fairly normal;" etc. For *2*:—"marked thickening of antero-inferior parts and moderate elsewhere;" "small calcified plaque, moderate thickening elsewhere;" etc.

Weber: This test was usually made with a 512 d.v. fork. Lateralization is shown by *R* or *L*, according to the ear that the sound was referred to; *n* = not lateralized.

BC time: This is usually for a 512 d.v. fork. The letter *n* means it was heard the normal time; *n* − means within the range of normal for the conditions of the test. (A column is not given for the Rinne tests, since for all cases in which it was possible to make fork tests air conduction was better than bone conduction.)

Middle-Ear Conditions: For the nine items under this heading the histologic observations are recorded on the general principle stated above. The lesions listed under *n* −, *1* and *2* consist for the most part of various degrees of increased fibrous tissue. See text in section on Middle Ear in Control Group. The ears with lesions rated as *3*, *4* or *5* are given special descriptions in the text for the respective groups. Blank spaces in this section of the tables indicate that the middle ear was broken in removal at autopsy and that the part is not present in the sections.

Ganglion Cells: See text, p. 330. For young adults with good hearing the total number (*Total No.*, given in thousands) averages 31,800; and the average number of ganglion cells corresponding to each mm. of length of the organ of Corti (*Ave. No. per Mm.*) is, in the lower basal turn, 1050, and in the upper basal 1350 (Dr. Hardy).

Blood Vessel Lesions: For the items of this section of the tables *n* means either normal or "no lesions found." The data for aorta, cerebral arteries and arteriolosclerosis are from the autopsy protocols; for the internal carotid artery they are from our observations of the part of this artery included in the temporal-bone sections (the space is left blank when this artery was torn out of the block removed at autopsy). The "plus" signs in the columns for arteries in the internal auditory meatus (*Int. Meatus*) and modiolar arteries mean that in one or more sections we observed small arteries with much thicker walls than normal for the size of the lumen; usually these changes are in only a small proportion of the vessels present. The "plus" signs in the column headed "*Temp. Bone Reg.*" record those cases in which definitely pathologic small vessels were observed in parts of the temporal bone sections other than the labyrinth proper.

TABLE 1

Data for 15 ears of the control group

All of these patients had good hearing for all tones up to and including 81

Ear Number	Autopsy Number / Sex / Race / Age	Side	Otoscopic	Weber	Middle Ear Conditions: Tym. / P. tensa / P. flaccida / Epitympanum / Ant.-Sup. Rec. / Malleus, Incus / Pos.-Sup. Rec. / Pos.-Inf. Rec. / Promontorium / Hyperplasia / Tinnitus	High Tone Acuity: Audiometer Readings 1024	2048	4096	8192	Histologic Observations of Conditions in the Basal Turns of the Cochlea: Nerve Fibers (Lower / Upper)	Organ of Corti (Lower / Upper)	Ext. Sulcus Cells (Lower / Upper)	Stria Vascularis (Lower / Upper)
													mm. 5 10 15	5 10 15	5 10 15	5 10 15
1	3832 R ♂ C 23	N	L N	L N	N N N N N N N N N	–	15	-5	0	-5						
2	3741 L ♂ C 37		N N	N N	N 1 N N N	–	15	10	5	5						
3	3832 L ♂ C 23	N	L N	L N	N N N	–	5	-5	10	-5						
4	4115 L ♀ C 25	N	R N	R N	N N N N N N N N N	–	10	10	10	0						
5	4340 R ♂ C 26	1	R N	R N	N N N N N N N N N	–	10	0	10	-5						
6	10891 L ♀ C 31	N			N N N N N N N N N	–	5	10	10	-5						
7	11787 R ♀ W 46	1	R N	R N	1 N N N N N 1 N N	–	5	10	15	0						
8	10715 R ♂ W 49	N	R N	R N	N N N N N N N N N	–	10	5	15	20						
9	10715 L ♂ W 49	N	R N	R N	1 N N N N N N N N	–	10	5	15	15						
10	3941 R ♂ C 37		R N	R N	N N N	–	15	10	15	-5						
11	4115 R ♀ C 25	N	R N	R N	N N N N N N N 1 1	–	20	10	15	5						
12	4340 L ♂ C 26	1	R N	R N	N N N N N N 1 N N	–	10	0	15	5						
13	10891 R ♀ C 31	N			N N N N N N N N N	–	5	10	15	-10						
14	11787 L ♀ W 46	1	N N	N N	1 N N N N N 1 N N	–	-5	-5	20	-5						
15	11157 R ♀ W 56	N	N	N	N N N N N N N N N	–	0	30	20	20						

TABLE 1

ata for 15 ears of the control group

good hearing for all tones up to and including 8192 d.v.

| Conditions in the Basal Turns of the Cochleae | | |

		Syphilis	Blood Pressure in mm. Hg.	Blood Vessel Lesions							Other - Inner Ear Lesions and Remarks	Ear Number
				Aorta	Int. Carotid	Cerebral Ar.	Arteriolarsel.	Int. Meatus	Modiolar Ar.	Temp. Bone		
	-			N	N	N	T	N	N	N		1
	+		125/65	+	+	N	N	N	N	N		2
	-			N	N	N	N	N	N	N		3
	-		98/65	N		N	N	N	N	N		4
	-		98/64	N	N	N	N	N	N	N		5
	-		190/140	+	+	+	+	N	N	+	Retinal Arteriosel.	6
	-		145/100		+			N	N	+	Papilledema	7
	-		142/112	+		N	N	N	N	N		8
	-		142/112	+ +	+	N	N	N	N	N		9
	+		125/65	+	+	N	N	N	N	N		10
	-		98/65	N		N	N	N	N	N		11
	-		98/64	N	N	N	N	N	N	N		12
	-		178/140	+	+	+	+	N	N	+	Retinal Arteriosel.	13
	-		145/100		+				N	N	Papilledema	14
	-		112/80		N		N	N	N			15

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TABLE 2

The data for the 24 ears of Group A of the "gradual" high-tone los

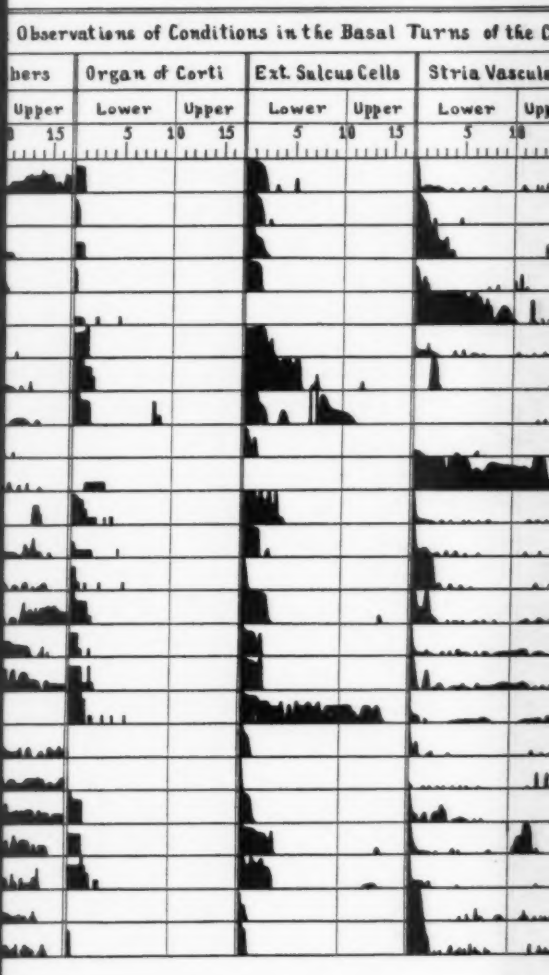
The principal lesion is partial nerve atrophy

Ear Number	Autopsy Number / Sex / Race / Age	Otoscopic: Side	Otoscopic: Rinne	Otoscopic: Weber	Middle Ear Conditions: T.T.	P. tensa	P. flaccida	Ext. meatus	Ant.-sup. Ret.	Malleus, lower	Fen. Oval. Ret.	Fen. Rot. Ret.	Promontory	Nuc. caudatus	Hyperacusis	Tinnitus	High Tone Acuity: Audiometer Readings 1024	2048	4096	8192	Histologic Observations of Conditions in the Basal Turns of the Cochlea: Nerve Fibers Lower	Nerve Fibers Upper	Organ of Corti Lower	Organ of Corti Upper	Ext. Sulcus Cells Lower	Ext. Sulcus Cells Upper	Stria Vascularis Lower	Stria Vascularis Upper
																					mm. 5	10	15	5	10	15	5	10	15	5	10	15	5	10	15	5	10	15
1	11748 R ♂ W 64				n	n	1	2	n	n	n	n	n	n	+	20	25	35	40																
2	11732 L ♀ C 59				n	2	n	2	n	n	n	n	n	n	−	15	20	35	55																
3	11732 R ♀ C 59				1	2	n	2	n	n	n	n	n	n	−	20	30	40	65																
4	10817 L ♂ W 35	n.			1	n	n	1	n	2	n	n	n	n	−	15	30	45	55																
5	4065 L ♀ C 40	n.			n	n	n	n	n	n	n	n	n	n	−	20	35	45	n.h.																
6	11932 L ♀ W 61	n.									n	n	n		−	5	35	50	n.h.																
7	3923 R ♂ W 70	n.			n	n	n	2	n	n	n	n	n	n	−	35	35	55	n.h.																
8	3923 L ♂ W 70	n.			1	n	1	1	n	n	1	n	n	n	−	30	35	55	n.h.																
9	3858 L ♂ W 39	n.	n.		n		1	1	n	1	n	n	n	n	−	25	45	60	45																
10	4065 R ♀ C 40	n.	n.		n	n	n	n	n	n	n	n	n	n	−	30	40	60	n.h.																
11	11932 R ♀ W 61	n.			n		n			n	n	n	n	n	−	20	35	60	n.h.																
12	10670 R ♀ W 63	1	n.	n.	n	n	1	1	n	n	n	n	n	n	−	30	55	60	70																
13	10670 L ♀ W 63	1	n.	n.			1	2	n	n	n	n	n	n	−	30	35	60	60																
14	11748 L ♂ W 64				n	n	1	1	n	n	n	n	n	n	+	15	25	60	60																
15	11983 R ♀ W 66	n.									n	n	n		+	30	35	60	65																
16	11983 L ♀ W 66	n.			n	n	n	1	n	n	n	n	n	n	+	40	30	65	65																
17	3725 L ♂ W 64	n.	n.		n	n	n	2	n	1	1	n	n	n	+	55	60	70	n.h.																
18	4359 R ♂ W 64	n.	L.		n	n	n	2	n	n	n	n	n	n	−	40	45	70	n.h.																
19	4359 L ♂ W 64	n.	L.		n	n	n	2	n	n	n	n			−	30	50	70	n.h.																
20	3781 R ♂ C 68	n.	L.	n.	n	n	n	1	n	1	n	n	n	n	−	40	30	70	60																
21	4111 L ♂ C 78	n.	L.								n	n	1	n	−	50	55	70	55																
22	4111 R ♂ C 78	n.	L.		n						n	n	n	n	+	45	55	70	n.h.																
23	4320 R ♂ W 66	n.	n.		n	n	1	2	1	1	1	1	n		+	35	45	80	n.h.																
24	4320 L ♂ W 66	n.	n.		n	n	n	2	n	n	n	n	n	n	−	30	60	85	n.h.																

TABLE 2

The data for the 24 ears of Group A of the "gradual" high-tone

The principal lesion is partial nerve atrophy



high-tone loss cases

trophy

of the Cochlea																			
a Vascularis		Bas. Memb.			Ganglion Cells								Blood Vessel Lesions						
ver	Upper	Calcif.	Ca.+Hy.	Monochoni-Ac.	Total No.	Ave. No. Per Mm. in Lower basal	Ave. No. Per Mm. in Upper basal	Systemic Diseases (Principal Diagnoses, Autopsy plus Clinical)	Syphilis	Blood Pressure in mm. Hg.	Aortic.	Int. Carotid	Cerebral Ar.	Arteriolarsc.	Int. Meatus	Basilar Ar.	Temp. Bone	Other Inner Ear Lesions and Remarks	Ear Number
5 10 15		mm.	mm.																
		0.0	1.5	14	24	850	870	Adrenal Tumor Arteriolarsci.	+	120/75	+	+	+	+			R	+	Slight Nerve Atr. Mid. turn	1
		0.7	2.7	14				Chr. Myocarditis	–	170/110	+	+	+	R	R	R	R	Retinal Artery	2
		0.0	1.2	13				Ditto	–	170/110	+		+	R		R	R	Ditto	3
		0.0	0.0	13				Rh. Endocarditis	–	150/70	+	+	R	R	R	R	R		4
		0.0	2.5	8	26	740	1010	Pulm. Tbc.	–	140/90	R	+	R	R	+	?	R	Sacc. Otolithic Memb. Les.	5
		0.3	1.2	13				Arteriolarsci. Nephritis, Chr.	–	200/120	+		R	+	R	R	+	Retinal Atrophy	6
		4.6	9.0	10				Arterioscleros. Nephritis, Chr.	–	170/80	+ +	+	+	R	+	?	R		7
		3.5	9.0	10				Ditto	–	170/80	+ +		+	R	+	?	R		8
		2.0	3.5	15	27	980	1210	Chr. Myocarditis Pulm. Arteriosci.	–			R	R	R	R	R	R		9
		0.0	1.2	9	28	780	1040	Pulm. Tbc.	–	140/80	R	+	R	R	+	R	R	Sac. Otolithic Memb. Swollen	10
		0.3	1.2	13				Arteriolarscler. Nephritis, Chr.	–	200/120	+	+	R	+	R	R	+	Retinal Atrophy	11
		0.3	0.3	11	28	715	1160	Diabetes mell. Arteriosclerosa.	+	160/74	+		+	R	R	R	+	Retinal Atrophy	12
		1.2	1.2	12	27	850	1180	Ditto	+	160/74	+		+	R	R	R	+	Ditto	13
		0.4	2.0	10	18	480	810	Adrenal Tumor Arteriolarsci.	+	120/75	+	+	+	+	R	R	+	Slight Nerve Atr. L. Mid. T.	14
		0.0	1.5					Arteriolarsci. Nephritis, Chr.	–	200/150	+ +		+	+	R	R	+	Partial Atr. Nu. to Cristae	15
		0.0	1.5					Ditto	–	200/150	+ +		+	+	R	R	+	Ditto	16
		0.0	2.0	12				Carc. Esoph. & mult. metast.	–	150/100	+	+ +	+	R	R	R	R	Metast. Tumor R. temp.-par.	17
		1.0	1.0	13				Pulm. Tbc. Pyelonephritis	–	95/25	+	+ +	+	R	R	R	R		18
		1.0	1.0	13				Ditto	–	95/25	+		+	R	R	R	R		19
		1.5	6.7	13	21	500	745	Syphilis, & Aortic Aneurism	+	140/105	+ +		R	R	R	R	R	A very long cochlea	20
		2.0	4.4	12				Arteriosclerosis Nephritis, Chr.	–	120/90	+ +	+ +	+	R		R	+		21
		2.0	4.4	13	17	340	840	Ditto	–	120/90	+ +	+ +	+	R	+	R	+		22
		1.0	5.3	12				Carc. Lung & mult. metast.	–	130/80	+	+ +	R	R	+	+	R		23
		1.0	3.5	11				Ditto	–	130/80	+	+ +	R	R	+	+	R		24

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TABLE 4

The data for the 17 ears of Group D of the "gradual" high-tone loss co

In none of these ears is the nerve atrophy more than in some ears of the c

Ear Number	Autopsy Number / Sex / Race / Age / Side	Otoscopic	Weber	Middle Ear Conditions: T. Tens. / P. Flaccidity / Exhaustion / Ant.-Sup. Reg. / Malleus, Body / Ext. Oval Reg. / Ext. Rot. Reg. / Promont. / Mes. Upp. Tym.	Tinnitus	High Tone Acuity: Audiometer Readings 1024	2048	4096	8192
30	11157 L ♀ W 56	N	N	N N N N N N N N N	–	0	25	20	30
31	10729 R ♀ W 67	N	N N	N N N 1 N N N N N	–	5	20	20	35
32	10729 L ♀ W 67	N	N N	N N 1 1 N N N N N	–	0	30	25	30
33	3764 R ♂ C 43	N	N N	N N N N N N N N N	+	15	10	30	40
34	11869 L ♀ W 61	N		N 1 N N N N N N N	–	25	25	30	40
35	11869 R ♀ W 61	N		N N N 1 N N 1 N N	–	20	25	35	65
36	10761 R ♀ W 35	N		1 1 N N N 1 N N N	–	15	30	45	50
37	4310 L ♀ C 46	N	N	N N N 2 N N N N N	–	20	30	45	40
38	10618 R ♀ W 46	1	L N	N 1 N N	+	15	35	45	40
39	4009 R ♀ C 28	N	N N	N N N N N N N N	–	30	30	50	55
40	10917 R ♂ W 35	N	N	N N N N N 1 N N N	–	20	40	50	35
41	11685 R ♂ W 47	N	N N	1 N 1 1 N N 1 N N	+	20	25	50	50
42	11415 R ♀ W 66	N	L N	N N N 1 1 N N N N	+	20	35	50	50
43	3838 R ♂ W 39	N	N N	1 N 1 1 N N 1 N N	–	20	40	55	65
44	4310 R ♀ C 46	N	N	N N N 2 N N N N N	–	25	30	55	50
45	11885 L ♂ W 47	N	N N	N 1 N N	+	20	25	60	50
46	11415 L ♀ W 66	N	L N	N N N 1 1 1 N N N	+	20	35	60	50

Histologic Observations of Conditions in the Basal Turns of the Co

Nerve Fibers: Lower	Nerve Fibers: Upper	Organ of Corti: Lower	Organ of Corti: Upper	Ext. Sulcus Cells: Lower	Ext. Sulcus Cells: Upper	Stria Vascula: Lower	Stria Vascula: Upp
mm. 5 10 15	5 10 15	5 10 15	5 10 15	5 10 15	5 10 15	5 10	

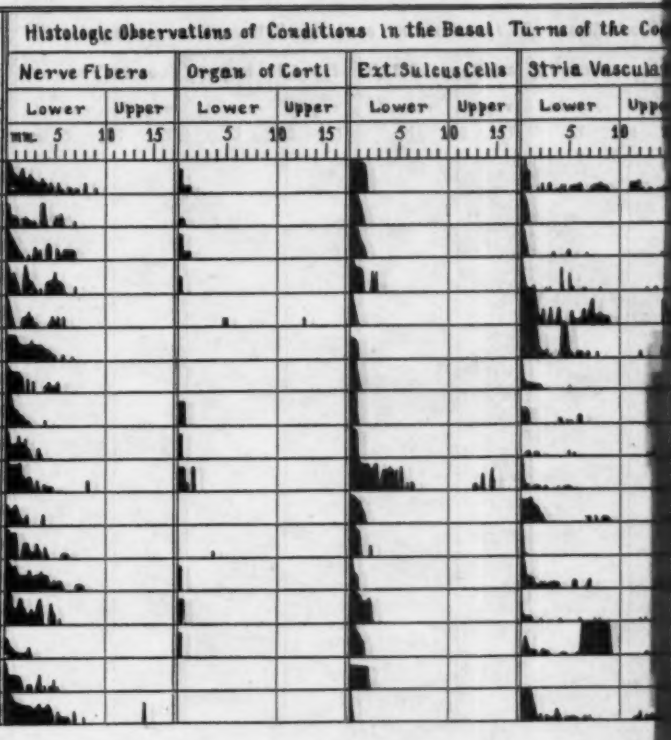
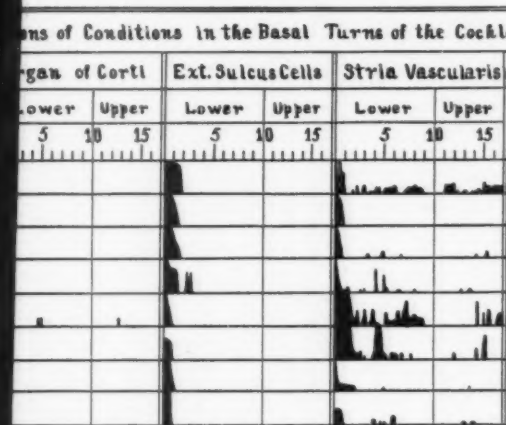


TABLE 4

r the 17 ears of Group D of the "gradual" high-tone loss cases

rs is the nerve atrophy more than in some ears of the control group



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Motion-Al.	Sytemic Diseases (Principal Diagnoses, Autopsy plus Clinical)	Syphilis	Blood Pressure in mm. Hg.	Blood Vessel Lesions: Aorta	Int. Carotid	Cerebral Ae.	Arteriolarscl.	Int. Meatus	Modiolar Ae.	Temp. Bone Ae.	Other Inner Ear Lesions and Remarks	Ear Number
3	Duv. Endothel-ioma, R. front.	–	152/80		+	N		N	N	N		30
2	Myocard. Insuf. Arteriosel.	–	200/110	+	+	+	N	N	N	N		31
6	Ditto	–	200/110	+	+	+	N	N	N	N		32
3	Syphilis of CNS. & Tabes dors.	+	136/70	N	+	N	N	N	N	N	2 Fibr. trab. in Sc. Vest.	33
4	Diabetes mell. Ac. Endocard.	–	150/85	+		+	N	N	N	N		34
4	Ditto	–	150/85	+		+	N	N	N	N	A Fibr. trab. in Sc. Vest.	35
6	RA. Endocard-itis, Chr.	–	150/70	N	N	N	N	N	N	N		36
3	Pulm. Tbc.	+	122/70	+	+	N	N		N	N	A Fibr. trab. in Sc. Vest.	37
3	Pineal Tumor	–	110/70	N		N	N	N	N	N		38
4	Pulm. Tbc.	–	99/70	N	+	N	N	N	N	N		39
3	R Rheum. Endocar.	–	130/70	+		N	N	N	N	+		40
4	Carc. Colon	–	126/76	+		N	N	N	N	N		41
2	RRheum. Endocard. Arteriolarscl.	–	210/120	+	±	+	+	N	N	N		42
3	Chr. Myocarditis Pulm. Arteriosel.	–		N		N	N	N	N	N		43
3	Pulm. Tbc.	+	123/78	+	+	N	N		N	N	A Fibr. trab. in Sc. Vest.	44
4	Carc. Colon	–	120/78	+		N	N	N	N	N		45
2	RA. Endocardit. Arteriolarscl.	–	214/120	+		+	+	N	N	N		46

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TABLE 5

The data for the 24 ears of Group E of the "abrupt" high-tone l

All have organ of Corti and external sulcus cell atrophy, in addition

Ear Number	Autopsy Number / Sex Race Age	Side	Otoscopic	Rinne / Weber	Middle Ear Conditions: Tym. Fibrosis	Tym. Retraction	Tym. Effusion	Tym. Perforation	Eustachian Obstruction	Ant.-Sup. Rec.	Malleus, Incus	Fen. Oval. Reg.	Fen. Rot. Reg.	Promontorium	Mucosa Hyperplasia	Tinnitus	High Tone Acuity: Audiometer Readings 1024	2048	4096	8192
47	11201 R ♂ W 32	1		R / R-	R	R	R	R	R	R	R-	R	R			–	5	15	15	45
48	11745 R ♂ W 42	R		R / R	1			1	1	R-	1	R	R			–	-5	5	25	60
49	11745 L ♂ W 42	R		R / R	R	1	R-	R-	1	R	1	R	R			–	5	5	40	70
50	10997 L ♂ W 46				R	2	2	R-	R	R	R-	R-	R	R		–	10	20	nk.	nk.
51	3792 R ♂ C 38	R		R / R	R	R	R	R	R	1	R-	R	R			–	15	15	45	55
52	3886 L ♂ W 60	1		R / R	1	R	R	R	R	R-	R-	R	R			–	20	25	65	nk.
53	3886 R ♂ W 60	1		R / R	1	R	R	1	R	2	1	R	R			–	15	20	75	nk.
54	3620 R ♂ C 38	R							R	R	R					+	20	25	80	nk.
55	3620 L ♂ C 38	R			R	R	R	R	R	R	R	R	R-			–	15	20	nk.	nk.
56	11992 L ♂ W 65	1		R / 36	1	R	R	2	R	R-	2	2				–	10	35	80	60
57	11992 R ♂ W 65	1		R / 55	1	R	R	1	R	R	1	R	R			–	15	40	60	nk.
58	11824 L ♂ W 26	R		R	Acute purulent otitis after testing											–	20	35	65	40
59	10620 L ♂ W 49	1		R	R	R	R	1	R	R	R	R	R			+	10	50	55	60
60	10620 R ♂ W 49	1		R	R	R	R	1	R	R	R-	R-	R	R		+	10	40	60	55
61	4108 R ♂ C 36	R		R / R-	R	2	R	1	R	R	R-	R-	R	R		+	15	30	65	50
62	3793 L ♂ W 71	R		R / R-	R	R	R	1	R	1	R	R	R			–	20	50	75	55
63	11082 L ♂ W 35	1			1				R-	R-	2	2				+ +	15	35	80	65
64	11082 R ♂ W 35	2			2	2	1	1	R-	2	1	2	2			+ +	10	30	85	75
65	4018 R ♂ W 41	R		R / R	R	R	R	R	2	R-	1	R	R			+	15	70	70	nk.
66	4478 L ♂ C 46	1		R	1	1	R	1	R-	R-	R-	R	R			–	25	70	70	nk.
67	3793 R ♂ W 71	R		R / R-	R	R	R	2	R	R	1	R	R			–	20	55	75	nk.
68	4501 L ♂ W 55	R		R	1	1	1	1	R	R-	R-	R	R			+	25	50	80	nk.
69	4479 R ♂ C 46	R		R	R	R	R	R-	R-	R-	R	R	R			–	20	60	nk.	nk.
70	4018 L ♂ W 41	R		R / R-	R			2	R-	1	R	R				+	20	70	nk.	nk.

Histologic Observations of Conditions in the Basal Turns of the Co

Nerve Fibers		Organ of Corti		Ext. Sulcus Cells		Stria Vascular	
Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upp
mm. 5 10 15		5 10 15		5 10 15		5 10	

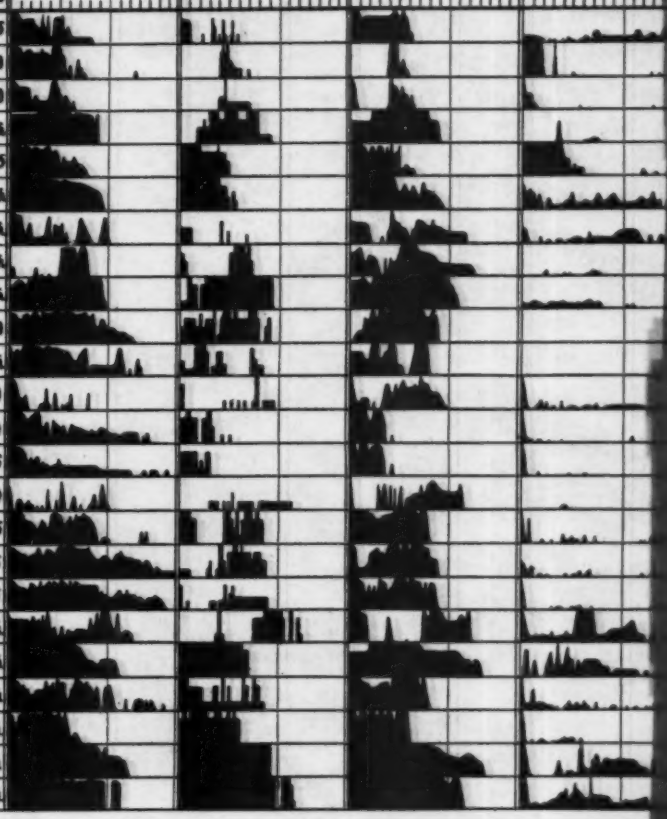
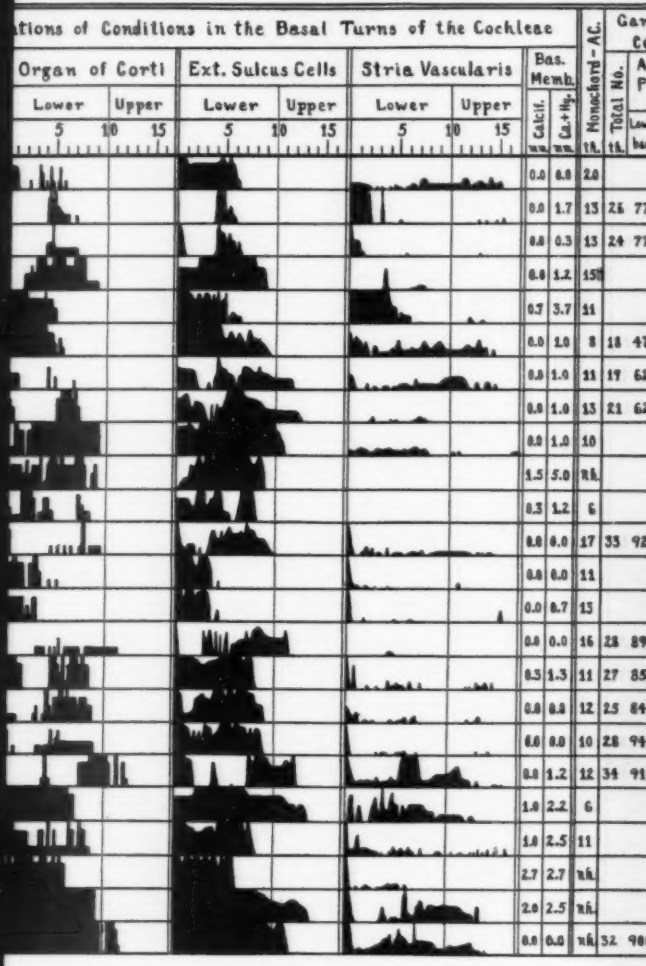


TABLE 5

The data for the 24 ears of Group E of the "abrupt" high-tone loss cases
organ of Corti and external sulcus cell atrophy, in addition to nerve atrophy



s

e atrophy

		Ganglion Cells			Systemic Diseases (Principal Diagnoses, Autopsy plus Clinical)	Syphilis	Blood Pressure in mm. Hg.	Blood Vessel Lesions						Other Inner Ear Lesions and Remarks	Ear Number
	Monochord - Ac. th.	Total No. th.	Ave. No. Per Mm. in Lower basal	Upper basal				Aorta	Int. Carotid	Cerebral Ac.	Articulatrpd.	Int. Acustica	Medialar Ac. Temp. Bone Rel.		
.0	20				Pituitary tumor & Diabetes insip.	-	95/65	N	+	N	N	N	N	Heard freq 5793 at 18	47
.7	13	26	770	980	Rt. Cerebal Glioma & Hemiplegia	-		+		N	N	+	N	5793 at 0	48
.3	13	24	770	1040	Ditto	-		+	+	N	N	+	N	5793 at 0	49
.2	15				Rheum. Endocard. Cirrh. of Liver	-	130/75	N	N	N	N	+	+	Plumbism (Rad) 1876 at 15	50
.7	11				Syphilis of CNS. & Tabes dors.	+	120/75	+	+	N	N	+	+		51
.0	8	18	470	840	Arteriosclerosis & Anaur. Cir. Willis	-	150/110	±	+	±	N	+	+		52
.0	11	17	620	880	Ditto	-	150/110	±	±	±	N	+	+		53
.0	13	21	620	860	Pneumonia	-	124/88	N		N	N	N	N		54
.0	10				Ditto	-	126/88	N		N	N	N	N		55
.0	nk.				Ca. Gall bladder & mult. metast.	-	162/98	+	+	N	N	+	+	Boilermaker 1446 at 15	56
.2	6				Ditto	-	162/98	+		N	N	+	+	Ditto	57
.0	17	33	920	1290	Calcinosis gen. Nephritis chr.	-	118/80	±	±	±	+	N	N	+	58
.0	11				Rheum. Endocard. Nephritis chr.	-	156/80	+	+	N	N	N	N		59
.7	13				Ditto	-	156/80	+	+	N	N	N	N		60
.0	16	25	890	1260	Pulm. Tbc.	±		N	N	N	N	N	N	3 Fibr. trab. in Sc. Vest.	61
.5	11	27	850	1220	Pulm. Tbc.	-	98/62	+	N	N	N	N	N		62
.8	12	25	840	1060	Hodgkin's dis.	-	110/66	N	N	N	N	+	+	N	63
.0	10	28	940	1220	Ditto	-	110/80	N	N	N	N	+	+	N	64
.2	12	34	910	1350	Carc. of Lung & mult. metast.	-	112/58	+	N	+	N	+	+	+	65
.2	6				Pulm. Tbc.	-		N	+	N	N	+	+	N	66
.5	11				Pulm. Tbc.	-	98/62	+		N	N	+	N	N	67
.7	nk.				Carc. Prostate Arteriosclerosis	-	135/85	+	+	+	N	+	+	+	Riveter for 15 yrs.	68
.5	nk.				Pulm. Tbc.	-		N	+	N	N	+	+	N		69
.0	nk.	32	980	1310	Carc. of Lung & mult. metast.	-	112/58	+	N	+	N		N	+	Atr. change in Limbus lb	70

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TABLE 3

The data for the ears of Groups B and C of the "gradual" high-tone

In each of these 5 ears there are middle-ear lesions more severe than in any

Ear Number	Autopsy Number / Sex Race Age	Otoscopic Side	Weber	Middle Ear Conditions: Bc time	P. tensa	P. flaccida	Epitympanum.	Ant.-Sup. Rec.	Malleus, Incus	Fen. Ovalis Rec.	Fen. Rot. Rec.	Promont.	Hypotym.	Mucous	Tinnitus	High Tone Acuity: Audiometer Readings 1024	2048	4096	8192
25	3867 L ♂ W 64	1	R N	2	3	1	2	N	3	2	N	1		—		30	20	40	55
26	3867 R ♂ W 64	2	R N	2	3	2	2	1	3	2	2	2		—		50	30	50	60
27	3725 R ♂ W 64	R	R R	1	N	N	2	3	3	1	N	N		—		50	55	55	n.h.
28	3728 R ♂ W 42	4	L N	5	5	N	2	3	N	N	1	N		—		35	40	45	50
29	3728 L ♂ W 42	3	L N	2	3	4	2	4	N	N	1	N		—		40	50	65	n.h.

Histologic Observations of Conditions in the Basal Turns of the Coc

Nerve Fibers — Lower / Upper; Organ of Corti — Lower / Upper; Ext. Sulcus Cells — Lower / Upper; Stria Vascularis — Lower / Upp

mm. 5 10 15 | 5 10 15 | 5 10 15 | 5 10

TABLE 6

The data for the ears of groups F, G and H of the "abrupt" high-ton

Ear Number	Autopsy Number / Sex Race Age	Otoscopic Side	Weber	Middle Ear Conditions: Bc time	P. tensa	P. flaccida	Epitympanum.	Ant.-Sup. Rec.	Malleus, Incus	Fen. Ovalis Rec.	Fen. Rot. Rec.	Promont.	Hypotym.	Mucous	Tinnitus	High Tone Acuity: Audiometer Readings 1024	2048	4096	8192
71	4176 R ♂ W 63	R	N N	R		N	N	N	N	N	N	N	N	—		45	20	70	55
72	4176 L ♂ W 63	N	R N	N		N	1	N	N	N	N	N	N	—		20	20	70	n.h.
73	10782 L ♂ W 48	N	R N	N	1	N	1	N	1	1	1	1		—		10	45	60	40
74	11928 R ♂ W 39	R		1	1	3	1	3	3	1	1	1		—		20	15	15	50
75	10782 R ♂ W 46	R	R R	2					3	2	1	1		—		5	5	40	55
76	4089 L ♀ C 28	N	N N	N	N	N	N	N	N	N	N	N		—		25	15	10	40
77	10791 R ♂ W 65		L N	N	N	N	N	N	N	2	1	N	N	—		0	20	45	60
78	10791 L ♂ W 65		L N	N	N	N		N	2	2	N	1		—		10	25	65	70
79	4100 L ♂ C 36	N	N N	N	1	N	1	N	N	N	N	N	N	+		15	15	45	n.h.

Histologic Observations of Conditions in the Basal Turns of t

Nerve Fibers — Lower / Upper; Organ of Corti — Lower / Upper; Ext. Sulcus Cells — Lower / Upper; Stria Ve — Lower

mm. 5 10 15 | 5 10 15 | 5 10 15 | 5

TABLE 3

data for the ears of Groups B and C of the "gradual" high-tone loss cases

e 5 ears there are middle-ear lesions more severe than in any of the control

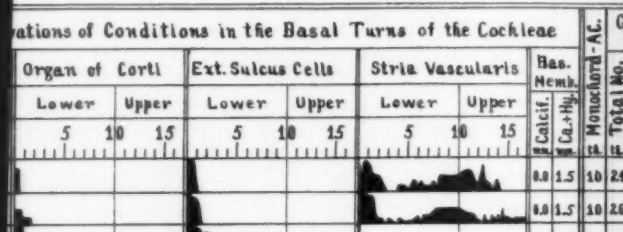
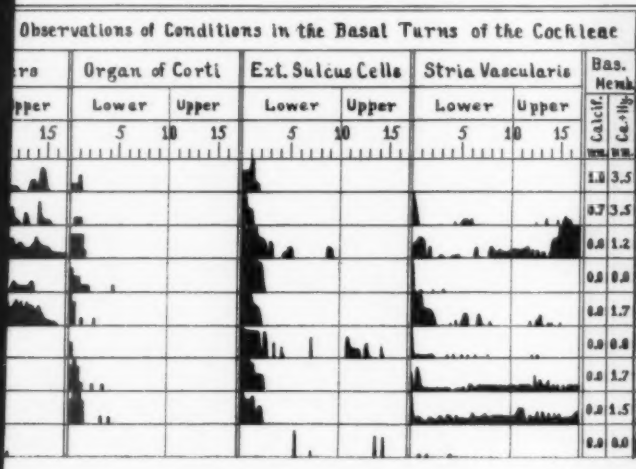


TABLE 6

data for the ears of groups F, G and H of the "abrupt" high-tone loss cases



cases

he control group

ae Bas. Memb. Calcif. mm.	Ca.+Hy. mm.	Monochord-AC. Ft.	Ganglion Cells Total No.	Ganglion Cells Ave. No. Per Mm. in Lower basal	Ganglion Cells Ave. No. Per Mm. in Upper basal	Sytemic Diseases (Principal Diagnoses, Autopsy plus Clinical)	Syphilis	Blood Pressure in mm. Hg.	Blood Vessel Lesions: Aorta	Int. Carotid	Cerebral Ar.	Arteriolarel.	Int. Meatus	Modiolar Ar.	Temp. Bone Sec.	Other Inner Ear Lesions and Remarks	Ear Number
.0	1.5	10	24	760	1010	Myocarditis, Chr. Arteriosclerosis	–	180/105	+	+	N	N	N	N	+		25
.0	1.5	10	26	780	1130	Ditto	–	180/105	+	+	N	N	+	+	+		26
.0	1.0	11				Carc. Esoph. & mult. metast.	–	150/100	+	+	N	N	N	N	N	Metast. tumor R. temp.-par.	27
.3	1.3		28	870	1025	Transv. Myelitis, 1914. Thoracic	–	135/80	N	+	N	N	N	N	N		28
.3	1.3		27	850	1020	Ditto	–	135/88	N	N	N	N	N	N	N	Reissner's memb. (Fig.34)	29

cases

ochleae ria er 15 Bas. Memb. Calcif. mm.	Ca.+Hy. mm.	Monochord-AC. Ft.	Systemic Diseases (Principal Diagnoses, Autopsy plus Clinical)	Syphilis	Blood Pressure in mm. Hg.	Blood Vessel Lesions: Aorta	Int. Carotid	Cerebral Ar.	Arteriolarel.	Int. Meatus	Modiolar Ar.	Temp. Bone Sec.	Other Inner Ear Lesions and Remarks	Ear Number
	1.0	3.5	14	Rheum. Endocard.	–	112/74	+	N	N	N	N	N	Tymp. Beleges Atrophy – 5 mm	71
	0.7	3.5	12	Ditto	–	112/74	+	N	N	N	N	N	Ditto	72
	0.0	1.2	14	Glioma, Left Cerebral	–		N		N	N	N	N	Heard freq. 1448 at 10	73
	0.0	0.0	14	Exophth. Goiter Nephr., Ac. form.	–	150/65	N	N	N	N	N	N	5793 at 10	74
	0.0	1.7	12	Glioma, Left Cerebral	–		N	+	N	N	N	N		75
	0.0	0.0	14	Pulm. Tbc.	–	99/78	N	N	N	N	N	N		76
	0.0	1.7	14	Cerebral Arteriosclerosis	–	190/100	+	+ + +	N	N	N	N	2896 at 15	77
	0.0	1.5	8	Ditto	–	190/100	+	+ + +	N	N	N	N		78
	0.0	0.0	14	Pulm. Tbc.	±		N	N	N	N		N	A. Fibr. trab. in Sc. Vest.	79

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BOOK REVIEWS

The Nervous Child at School. By HECTOR CHARLES CAMERON. 160 pp. $1.50. (*Oxford Univ. Press, New York, 1933.*)

It is with an unusual maturity of judgment that Dr. Cameron discusses the problems of childhood associated with school life. While the ill effects of mental and bodily fatigue form the central theme of the book, other common disturbances are considered in a very understandable and practical way. It is likely that this small volume will attain a more important and permanent place in the literature of child guidance than its predecessor "The Nervous Child."

E. M. B.

Medicine in Canada. [Clio Medica.] By WILLIAM BOYMAN HOWELL. 1933. 137 pp. $1.50. (*Paul B. Hoeber, Inc., New York, 1933.*)

This chronological synopsis, well embroidered with episodic studies of the development of Canadian Medicine, as complete and compact as one could wish, covers an important and an often neglected development on the largest part of our continent. W. B. Howell has contributed in no small measure to the popularity of the Clio Medica series. They are those worthwhile "small books."

M. P.

California's Medical Story. By HENRY HARRIS. [With an Introduction by Charles Singer.] 421 pp. (*J. W. Stacey, Inc., San Francisco, California, 1932.*)

A bit tedious, and of merely remote interest to the ordinary student, the author's place is, nevertheless, of broad scope. He has taken great pains to list and record the minutiae of California's one hundred and sixty years existence under the rule of the white man. To the Californian this local history, which retains much of the pioneer interest, should be a most pleasing contribution.

M. P.

Dosage Tables for Röntgen Therapy. By PROFESSOR FRIEDRICH VOLTZ. [Translated from the 2nd German Edition.] 120 pp. $2.50. (*Oxford Univ. Press, New York, 1930.*)

A convenient reference in deciding upon X-ray dosage.

W. B. F.

The Chest. [Annals of Roentgenology, Vol. XI.] By L. R. SANTE. Illust. 561 pp. (*Paul B. Hoeber, Inc., New York, 1930.*)

The outstanding textbook on the Roentgen diagnosis of diseases of the chest.

A tendency to repetition in the various subdivisions serves merely to impress certain salient facts upon the reader's mind.

W. B. F.

The Modern Treatment of Syphilis. By JOSEPH EARLE MOORE, M.D. 535 pp. $5.00 (*Charles C. Thomas, Springfield, Ill., 1933.*)

"During the years between 1905 and 1910 the causative agent of syphilis was demonstrated, the Wassermann reaction was developed, and salvarsan was introduced. The first two contributions have greatly simplified the accurate diagnosis of this disease. The discovery of salvarsan marked the beginning of the modern treatment of syphilis."

Under this title the author has presented the experience of the past 25 years in the Syphilis Division of the Medical Clinic of the Johns Hopkins Hospital. In the space of 475 pages he has set forth an enormous amount of information. Moreover, he has clearly recognized the fact that observations upon the effects of treatment are, at the same time, studies upon the biology of the disease.

Following a succinct discussion of the biology and the prognosis of the disease, treated and untreated, the therapeutic problem is clearly formulated. The various antisyphilitic drugs, and the methods of their administration are then described in detail. The reviewer is impressed by the commendable caution indicated in devoting, page for page, as much space to the description of the toxic manifestations of the arsenical preparations and heavy metals, and the treatment thereof, as to the consideration of their antisyphilitic effects.

In the remaining, major portion of the book specific suggestions regarding treatment of the various stages and manifestations of the disease are fortified by statistical accounts of the therapeutic results in large numbers of cases. Special chapters are devoted to the treatment of visceral, cardiovascular, ocular and neural syphilis, to the management of syphilitic patients suffering from other, non-syphilitic, diseases, and to the management of syphilis in pregnancy. Here the reader will find a thorough, but not too dogmatic, discussion of the latest methods and results of treatment of almost any form of syphilis. Important practical details are given due consideration but the pages are not cluttered with hypotheses or theories. The sub-headings in each chapter add greatly to the clarity of the discussion. The bibliography is ample and well arranged.

Throughout the book the author appears to have considered from almost every angle the problem of how to explain the disease to the patient, and the, sometimes perplexing, social complications thereof. Particularly is this true in the final chapter, in which he gives counsel to the physician who is called upon to advise the syphilitic patient regarding marriage. Here the problem is squarely faced and a sane solution is offered.

Dr. Moore is to be complimented highly upon this carefully written treatise. The book itself is to be recommended to all physicians.

E. C. A.